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Francisco, CA 94131 (US).(54) Title: METHODS OF DIAGNOSIS OF OVARIAN CANCER, COMPOSITIONS AND METHODS OF SCREENING FOR
MODULATORS OF OVARIAN CANCER

(57) Abstract: Described herein are genes whose expression are up-regulated or down-regulated in ovarian cancer. Related methods and compositions that can be used for diagnosis and treatment of ovarian cancer are disclosed. Also described herein are methods that can be used to identify modulators of ovarian cancer.

METHODS OF DIAGNOSIS OF OVARIAN CANCER, COMPOSITIONS AND
METHODS OF SCREENING FOR MODULATORS OF OVARIAN CANCER

CROSS-REFERENCES TO RELATED APPLICATIONS.

5 This application is related to USSN 60/299,234, filed June 18, 2001; USSN 60/315,287, filed August 27, 2001; USSN 60/317,544, filed September 5, 2001; USSN 60/350,666, filed November 13, 2001; and USSN 60/372,246, filed April 12, 2002, each of which is incorporated herein by reference for all purposes.

10 FIELD OF THE INVENTION

The invention relates to the identification of nucleic acid and protein expression profiles and nucleic acids, products, and antibodies thereto that are involved in ovarian cancer; and to the use of such expression profiles and compositions in the diagnosis, prognosis, and therapy of ovarian cancer. The invention further relates to methods for identifying and using agents and/or targets that inhibit ovarian cancer.

BACKGROUND OF THE INVENTION

Ovarian cancer is the sixth most common cancer in women, accounting for 6% of all female cancers. It ranks fifth as the cause of cancer death in women. The American Cancer Society predicts that there will be about 23,100 new cases of ovarian cancer in this country in the year 2000 and about 14,000 women will die of the disease. Because many ovarian cancers cannot be detected early in their development, they account for a disproportionate number of fatal cancers, being responsible for almost half the deaths from cancer of the female genital tract; more deaths than any other reproductive organ cancer.

25 Most patients with epithelial ovarian cancer, the predominant form, are asymptomatic in early-stage disease and usually present with stage III or IV disease. Their five-year survival is less than 25%, with lower survival among African-American women. The minority of patients discovered with early-stage disease have a five-year survival rate of 80%-90%. See, Parker, et. al. (1997) "Cancer Statistics, 1997" CA Cancer J.Clin. 47:5-27.

30 In the absence of a family history of ovarian cancer, lifetime risk of ovarian cancer is 1/70. Risk factors include familial cancer syndromes (risk of up to 82% by age 70 in women

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with hereditary breast/ovarian syndrome); family history (1.4% lifetime risk with no affected relatives, 5% with one affected relative, 7% with two affected relatives; Kerlikowski, et al. (1992) Obstet. Gynecol. 80:700-707); nulliparity; advancing age; obesity; personal history of breast, endometrial, or colorectal cancer; fewer pregnancies; or older age (>35 years) at first pregnancy. However, 95% of all ovarian cancers occur in women without risk factors. Use of hormonal contraceptives, oophorectomy, and tubal sterilization reduce risk of ovarian cancer (Kerlikowski, et al. (1992) Obstet. Gynecol. 80:700-707; Grimes (1992) Am J. Obstet. Gynecol. 166:1950-1954; Hankinson, et al. (1993) JAMA 270:2813-2818); however, even bilateral oophorectomy may not be completely effective in preventing ovarian cancer.

10 Treatment of ovarian cancer consists largely of surgical oophorectomy, anti-hormone therapy, and/or chemotherapy. Although many ovarian cancer patients are effectively treated, the current therapies can all induce serious side effects which diminish quality of life. Deciding on a particular course of treatment is typically based on a variety of prognostic parameters and markers (Fitzgibbons, et al. (2000) Arch. Pathol. Lab. Med. 124:966-978; Hamilton and Piccart (2000) Ann. Oncol. 11:647-663), including genetic predisposition markers BRCA-1 and BRCA-2 (Robson (2000) J. Clin. Oncol. 18:113sup-118sup).

The identification of novel therapeutic targets and diagnostic markers is essential for improving the current treatment of ovarian cancer patients. Recent advances in molecular medicine have increased the interest in tumor-specific cell surface antigens that could serve as targets for various immunotherapeutic or small molecule strategies. Antigens suitable for immunotherapeutic strategies should be highly expressed in cancer tissues and ideally not expressed in normal adult tissues. Expression in tissues that are dispensable for life, however, may be tolerated. Examples of such antigens include Her2/neu and the B-cell antigen CD20. Humanized monoclonal antibodies directed to Her2/neu (Herceptin®/trastuzumab) are currently in use for the treatment of metastatic breast cancer. Ross and Fletcher (1998) Stem Cells 16:413-428. Similarly, anti-CD20 monoclonal antibodies (Rituxin®/rituximab) are used to effectively treat non-Hodgkin's lymphoma. Maloney, et al. (1997) Blood 90:2188-2195; Leget and Czuczman (1998) Curr. Opin. Oncol. 10:548-551.

30 Potential immunotherapeutic targets have been identified for ovarian cancer. One such target is polymorphic epithelial mucin (MUC1). MUC1 is a transmembrane protein, present at the apical surface of glandular epithelial cells. It is often overexpressed in ovarian cancer, and typically exhibits an altered glycosylation pattern, resulting in an antigenically

distinct molecule, and is in early clinical trials as a vaccine target. Gilewski, et al. (2000) Clin. Cancer Res. 6:1693-1701; Scholl, et al. (2000) J. Immunother. 23:570-580. The tumor-expressed protein is often cleaved into the circulation, where it is detectable as the tumor marker, CA 15-3. See, e.g., Bon, et al. (1997) Clin. Chem. 43:585-593. However, many patients have tumors that express neither HER2 nor MUC-1; therefore, it is clear that other targets need to be identified to manage localized and metastatic disease.

5 Mutations in both BRCA1 and BRCA2 are associated with increased susceptibility to ovarian cancer. Mutations in BRCA1 occur in approximately 5 percent (95 percent confidence interval, 3 to 8 percent) of women in whom ovarian cancer is diagnosed before the age of 70 years. See Stratton, et al. (1997) N.E.J. Med. 336:1125-1130. And, in BRCA1 gene carriers, the risk for developing ovarian cancer is .63. See Easton (1995) Am. J. Hum. Genet. 56:267-xxx; and Elit (2001) Can. Fam. Physician 47:778-84.

10 Other biochemical markers such as CA125 have been reported to be associated with ovarian cancer, but they are not absolute indicators of disease. Although roughly 85% of women with clinically apparent ovarian cancer have increased levels of CA125, CA125 is also increased during the first trimester of pregnancy, during menstruation, in the presence of non-cancerous illnesses, and in cancers of other sites.

While industry and academia have identified novel gene sequences, there has not been an equal effort exerted to identify the function of these novel sequences. The elucidation of a role for novel proteins and compounds in disease states for identification of therapeutic targets and diagnostic markers is essential for improving the current treatment of ovarian cancer patients. Accordingly, provided herein are molecular targets for therapeutic intervention in ovarian and other cancers. Additionally, provided herein are methods that can be used in diagnosis and prognosis of ovarian cancer. Further provided are methods that can be used to screen candidate bioactive agents for the ability to modulate ovarian cancer.

SUMMARY OF THE INVENTION

The present invention therefore provides nucleotide sequences of genes that are up- and down-regulated in ovarian cancer cells. Such genes are useful for diagnostic purposes, and also as targets for screening for therapeutic compounds that modulate ovarian cancer, such as hormones or antibodies. The methods of detecting nucleic acids of the invention or their encoded proteins can be used for many purposes, e.g., early detection of ovarian cancers, monitoring and early detection of relapse following treatment, monitoring response to therapy, selecting patients for postoperative chemotherapy or radiation therapy, selecting therapy, determining tumor prognosis, treatment, or response to treatment (of primary or metastatic tumors), and early detection of pre-cancerous lesions. Other aspects of the invention will become apparent to the skilled artisan by the following description of the invention.

In one aspect, the present invention provides a method of detecting an ovarian cancer-associated transcript in a cell from a patient, the method comprising contacting a biological sample from the patient with a polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26.

In one embodiment, the present invention provides a method of determining the level of an ovarian cancer associated transcript in a cell from a patient.

In one embodiment, the present invention provides a method of detecting an ovarian cancer-associated transcript in a cell from a patient, the method comprising contacting a biological sample from the patient with a polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26.

In one embodiment, the polynucleotide selectively hybridizes to a sequence at least 95% identical to a sequence as shown in Tables 1-26.

In one embodiment, the biological sample is a tissue sample. In another embodiment, the biological sample comprises isolated nucleic acids, e.g., mRNA.

In one embodiment, the polynucleotide is labeled, e.g., with a fluorescent label.

In one embodiment, the polynucleotide is immobilized on a solid surface.

In one embodiment, the patient is undergoing a therapeutic regimen to treat ovarian cancer. In another embodiment, the patient is suspected of having metastatic ovarian cancer.

In one embodiment, the patient is a human.

In one embodiment, the ovarian cancer associated transcript is mRNA.

In one embodiment, the method further comprises the step of amplifying nucleic acids

before the step of contacting the biological sample with the polynucleotide.

In another aspect, the present invention provides a method of monitoring the efficacy of a therapeutic treatment of ovarian cancer, the method comprising the steps of: (i) providing a biological sample from a patient undergoing the therapeutic treatment; and (ii) determining the level of an ovarian cancer-associated transcript in the biological sample by contacting the biological sample with a polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26, thereby monitoring the efficacy of the therapy. In a further embodiment, the patient has metastatic ovarian cancer. In a further embodiment, the patient has a drug resistant form of ovarian cancer.

In one embodiment, the method further comprises the step of: (iii) comparing the level of the ovarian cancer-associated transcript to a level of the ovarian cancer-associated transcript in a biological sample from the patient prior to, or earlier in, the therapeutic treatment.

Additionally, provided herein is a method of evaluating the effect of a candidate ovarian cancer drug comprising administering the drug to a patient and removing a cell sample from the patient. The expression profile of the cell is then determined. This method may further comprise comparing the expression profile to an expression profile of a healthy individual. In a preferred embodiment, said expression profile includes a gene of Tables 1-26.

In one aspect, the present invention provides an isolated nucleic acid molecule consisting of a polynucleotide sequence as shown in Tables 1-26.

In one embodiment, an expression vector or cell comprises the isolated nucleic acid.

In one aspect, the present invention provides an isolated polypeptide which is encoded by a nucleic acid molecule having polynucleotide sequence as shown in Tables 1-26.

In another aspect, the present invention provides an antibody that specifically binds to an isolated polypeptide which is encoded by a nucleic acid molecule having polynucleotide sequence as shown in Tables 1-26.

In one embodiment, the antibody is conjugated to an effector component, e.g., a fluorescent label, a radioisotope or a cytotoxic chemical.

In one embodiment, the antibody is an antibody fragment. In another embodiment, the antibody is humanized.

In one aspect, the present invention provides a method of detecting an ovarian cancer cell in a biological sample from a patient, the method comprising contacting the biological

sample with an antibody as described herein.

In another aspect, the present invention provides a method of detecting antibodies specific to ovarian cancer in a patient, the method comprising contacting a biological sample from the patient with a polypeptide encoded by a nucleic acid comprising a sequence from Tables 1-26.

5 In another aspect, the present invention provides a method for identifying a compound that modulates an ovarian cancer-associated polypeptide, the method comprising the steps of: (i) contacting the compound with an ovarian cancer-associated polypeptide, the polypeptide encoded by a polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26; and (ii) determining the functional effect of the compound upon the polypeptide.

In one embodiment, the functional effect is a physical effect, an enzymatic effect, or a chemical effect.

In one embodiment, the polypeptide is expressed in a eukaryotic host cell or cell

15 membrane. In another embodiment, the polypeptide is recombinant.

In one embodiment, the functional effect is determined by measuring ligand binding to the polypeptide.

In another aspect, the present invention provides a method of inhibiting proliferation of an ovarian cancer-associated cell to treat ovarian cancer in a patient, the method comprising the step of administering to the subject a therapeutically effective amount of a compound identified as described herein.

In one embodiment, the compound is an antibody.

25 In another aspect, the present invention provides a drug screening assay comprising the steps of: (i) administering a test compound to a mammal having ovarian cancer or to a cell sample isolated from; (ii) comparing the level of gene expression of a polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26 in a treated cell or mammal with the level of gene expression of the polynucleotide in a control cell sample or mammal, wherein a test compound that modulates the level of expression of the polynucleotide is a candidate for the treatment of ovarian cancer.

30 In one embodiment, the control is a mammal with ovarian cancer or a cell sample that has not been treated with the test compound. In another embodiment, the control is a normal cell or mammal, or is non-malignant tissue.

In one embodiment, the test compound is administered in varying amounts or

concentrations. In another embodiment, the test compound is administered for varying time periods. In another embodiment, the comparison can occur after addition or removal of the drug candidate.

5 In one embodiment, the levels of a plurality of polynucleotides that selectively hybridize to a sequence at least 80% identical to a sequence as shown in Tables 1-26 are individually compared to their respective levels in a control cell sample or mammal. In a preferred embodiment the plurality of polynucleotides is from three to ten.

In another aspect, the present invention provides a method for treating a mammal having ovarian cancer comprising administering a compound identified by the assay described herein.

10 In another aspect, the present invention provides a pharmaceutical composition for treating a mammal having ovarian cancer, the composition comprising a compound identified by the assay described herein and a physiologically acceptable excipient.

In one aspect, the present invention provides a method of screening drug candidates by providing a cell expressing a gene that is up- and down-regulated as in an ovarian cancer.

15 In one embodiment, a gene is selected from Tables 1-26. The method further includes adding a drug candidate to the cell and determining the effect of the drug candidate on the expression of the expression profile gene.

20 In one embodiment, the method of screening drug candidates includes comparing the level of expression in the absence of the drug candidate to the level of expression in the presence of the drug candidate, wherein the concentration of the drug candidate can vary when present, and wherein the comparison can occur after addition or removal of the drug candidate. In a preferred embodiment, the cell expresses at least two expression profile genes. The profile genes may show an increase or decrease.

25 Also provided is a method of evaluating the effect of a candidate ovarian cancer drug comprising administering the drug to a transgenic animal expressing or over-expressing the ovarian cancer modulatory protein, or an animal lacking the ovarian cancer modulatory protein, for example as a result of a gene knockout.

30 Moreover, provided herein is a biochip comprising one or more nucleic acid segments of Tables 1-26, wherein the biochip comprises fewer than 1000 nucleic acid probes.

Preferably, at least two nucleic acid segments are included. More preferably, at least three nucleic acid segments are included.

Furthermore, a method of diagnosing a disorder associated with ovarian cancer is

provided. The method comprises determining the expression of a gene of Tables 1-26 in a first tissue type of a first individual, and comparing the distribution to the expression of the gene from a second normal tissue type from the first individual or a second unaffected individual. A difference in the expression indicates that the first individual has a disorder associated with ovarian cancer.

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In a further embodiment, the biochip also includes a polynucleotide sequence of a gene that is not up- and down-regulated in ovarian cancer.

In one embodiment a method for screening for a bioactive agent capable of interfering with the binding of an ovarian cancer modulating protein (ovarian cancer modulatory protein) or a fragment thereof and an antibody which binds to said ovarian cancer modulatory protein or fragment thereof. In a preferred embodiment, the method comprises combining an ovarian cancer modulatory protein or fragment thereof, a candidate bioactive agent and an antibody which binds to said ovarian cancer modulatory protein or fragment thereof. The method further includes determining the binding of said ovarian cancer modulatory protein or fragment thereof and said antibody. Wherein there is a change in binding, an agent is identified as an interfering agent. The interfering agent can be an agonist or an antagonist. Preferably, the agent inhibits ovarian cancer.

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Also provided herein are methods of eliciting an immune response in an individual. In one embodiment a method provided herein comprises administering to an individual a composition comprising an ovarian cancer modulating protein, or a fragment thereof. In another embodiment, the protein is encoded by a nucleic acid selected from those of Tables 1-26.

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Further provided herein are compositions capable of eliciting an immune response in an individual. In one embodiment, a composition provided herein comprises an ovarian cancer modulating protein, preferably encoded by a nucleic acid of Table 1-26 or a fragment thereof, and a pharmaceutically acceptable carrier. In another embodiment, said composition comprises a nucleic acid comprising a sequence encoding an ovarian cancer modulating protein, preferably selected from the nucleic acids of Tables 1-26, and a pharmaceutically acceptable carrier.

25

Also provided are methods of neutralizing the effect of an ovarian cancer protein, or a fragment thereof, comprising contacting an agent specific for said protein with said protein in an amount sufficient to effect neutralization. In another embodiment, the protein is encoded by a nucleic acid selected from those of Tables 1-26.

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In another aspect of the invention, a method of treating an individual for ovarian cancer is provided. In one embodiment, the method comprises administering to said individual an inhibitor of an ovarian cancer modulating protein. In another embodiment, the method comprises administering to a patient having ovarian cancer an antibody to an ovarian cancer modulating protein conjugated to a therapeutic moiety. Such a therapeutic moiety can be a cytotoxic agent or a radioisotope.

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DETAILED DESCRIPTION OF THE INVENTION

In accordance with the objects outlined above, the present invention provides novel methods for diagnosis and prognosis evaluation for ovarian cancer (OC), including metastatic ovarian cancer, as well as methods for screening for compositions which modulate ovarian cancer. Also provided are methods for treating ovarian cancer and related conditions, e.g., ovarian carcinoma (e.g., epithelial (including malignant serous tumors, malignant mucinous tumors, and malignant endometrioid tumors), germ cell (including teratomas, choriocarcinomas, polyembryomas, embryonal carcinoma, endodermal sinus tumor, dysgerminoma, and gonadoblastoma), and stromal carcinomas (e.g., granulosa stromal cell tumors)), fallopian tube carcinoma, and peritoneal carcinoma.

Tables 1-26 provide unigene cluster identification numbers for the nucleotide sequence of genes that exhibit increased or decreased expression in ovarian cancer samples. Tables 1-26 also provide an exemplar accession number that provides a nucleotide sequence that is part of the unigene cluster.

Definitions

The term "ovarian cancer protein" or "ovarian cancer polynucleotide" or "ovarian cancer-associated transcript" refers to nucleic acid and polypeptide polymorphic variants, alleles, mutants, and interspecies homologues that: (1) have a nucleotide sequence that has greater than about 60% nucleotide sequence identity, 65%, 70%, 75%, 80%, 85%, 90%, preferably 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, or 99% or greater nucleotide sequence identity, preferably over a region of at least about 25, 50, 100, 200, 500, 1000, or more nucleotides, to a nucleotide sequence of or associated with a gene of Tables 1-26; (2) bind to antibodies, e.g., polyclonal antibodies, raised against an immunogen comprising an amino acid sequence encoded by a nucleotide sequence of or associated with a gene of Tables 1-26, and conservatively modified variants thereof; (3) specifically hybridize under stringent hybridization conditions to a nucleic acid sequence, or the complement thereof of Tables 1-26 and conservatively modified variants thereof; or (4) have an amino acid sequence that has greater than about 60% amino acid sequence identity, 65%, 70%, 75%, 80%, 85%, 90%, preferably 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, or 99% or greater amino sequence identity, preferably over a region of over a region of at least about 25, 50, 100, 200, 500, 1000, or more amino acid, to an amino acid sequence encoded by a nucleotide sequence of or associated with a gene of Tables 1-26. A polynucleotide or polypeptide

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sequence is typically from a mammal including, but not limited to, primate, e.g., human; rodent, e.g., rat, mouse, hamster, cow, pig, horse, sheep, or other mammal. An "ovarian cancer polypeptide" and an "ovarian cancer polynucleotide," include both naturally occurring or recombinant forms.

5 A "full length" ovarian cancer protein or nucleic acid refers to an ovarian cancer polypeptide or polynucleotide sequence, or a variant thereof, that contains all of the elements normally contained in one or more naturally occurring, wild type ovarian cancer polynucleotide or polypeptide sequences. The "full length" may be prior to, or after, various stages of post-translation processing or splicing, including alternative splicing.

10 "Biological sample" as used herein is a sample of biological tissue or fluid that contains nucleic acids or polypeptides, e.g., of an ovarian cancer protein, polynucleotide or transcript. Such samples include, but are not limited to, tissue isolated from primates, e.g., humans, or rodents, e.g., mice, and rats. Biological samples may also include sections of tissues such as biopsy and autopsy samples, frozen sections taken for histologic purposes, blood, plasma, serum, sputum, stool, tears, mucus, hair, skin, etc. Biological samples also include explants and primary and/or transformed cell cultures derived from patient tissues. A biological sample is typically obtained from a eukaryotic organism, most preferably a mammal such as a primate e.g., chimpanzee or human; cow; dog; cat; a rodent, e.g., guinea pig, rat, mouse, rabbit; or a bird, reptile; or fish. Livestock and domestic animals are of particular interest.

20 "Providing a biological sample" means to obtain a biological sample for use in methods described in this invention. Most often, this will be done by removing a sample of cells from an animal, but can also be accomplished by using previously isolated cells (e.g., isolated by another person, at another time, and/or for another purpose), or by performing the methods of the invention in vivo. Archival tissues, having treatment or outcome history, will be particularly useful.

25 The terms "identical" or percent "identity," in the context of two or more nucleic acids or polypeptide sequences, refer to two or more sequences or subsequences that are the same or have a specified percentage of amino acid residues or nucleotides that are the same (e.g., about 60% identity, preferably 70%, 75%, 80%, 85%, 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99%, or higher identity over a specified region, when compared and aligned for maximum correspondence over a comparison window or designated region) as measured using a BLAST or BLAST 2.0 sequence comparison algorithms with default

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parameters described below, or by manual alignment and visual inspection (see, e.g., NCBI web site <http://www.ncbi.nlm.nih.gov/BLAST/> or the like). Such sequences are then said to be "substantially identical." This definition also refers to, or may be applied to, the compliment of a test sequence. The definition also includes sequences that have deletions and/or additions, as well as those that have substitutions, as well as naturally occurring, e.g., polymorphic or allelic variants, and man-made variants. As described below, the preferred algorithms can account for gaps and the like. Preferably, identity exists over a region that is at least about 25 amino acids or nucleotides in length, or more preferably over a region that is 50-100 amino acids or nucleotides in length.

For sequence comparison, typically one sequence acts as a reference sequence, to which test sequences are compared. When using a sequence comparison algorithm, test and reference sequences are entered into a computer, subsequence coordinates are designated, if necessary, and sequence alignment program parameters are designated. Preferably, default program parameters can be used, or alternative parameters can be designated. The sequence comparison algorithm then calculates the percent sequence identities for the test sequences relative to the reference sequence, based on the program parameters.

A "comparison window", as used herein, includes reference to a segment of one of the number of contiguous positions selected from the group consisting typically of from 20 to 600, usually about 50 to about 200, more usually about 100 to about 150 in which a sequence may be compared to a reference sequence of the same number of contiguous positions after the two sequences are optimally aligned. Methods of alignment of sequences for comparison are well-known in the art. Optimal alignment of sequences for comparison can be conducted, e.g., by the local homology algorithm of Smith and Waterman (1981) Adv. Appl. Math. 2:482-489, by the homology alignment algorithm of Needleman and Wunsch (1970) J. Mol. Biol. 48:443-453, by the search for similarity method of Pearson and Lipman (1988) Proc. Nat'l. Acad. Sci. USA 85:2444-2448, by computerized implementations of these algorithms (GAP, BESTFIT, FASTA, and TFASTA in the Wisconsin Genetics Software Package, Genetics Computer Group, 575 Science Dr., Madison, WI), or by manual alignment and visual inspection (see, e.g., Ausubel, et al. (eds. 1995 and supplements) Current Protocols in Molecular Biology Lippincott).

Preferred examples of algorithms that are suitable for determining percent sequence identity and sequence similarity include the BLAST and BLAST 2.0 algorithms, which are described in Altschul, et al. (1977) Nuc. Acids Res. 25:3389-3402 and Altschul, et al. (1990)

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J. Mol. Biol. 215:403-410. BLAST and BLAST 2.0 are used, with the parameters described herein, to determine percent sequence identity for the nucleic acids and proteins of the invention. Software for performing BLAST analyses is publicly available through the National Center for Biotechnology Information (<http://www.ncbi.nlm.nih.gov/>). This

algorithm involves first identifying high scoring sequence pairs (HSPs) by identifying short words of length W in the query sequence, which either match or satisfy some positive-valued threshold score T when aligned with a word of the same length in a database sequence. T is referred to as the neighborhood word score threshold (Altschul, et al., supra). These initial neighborhood word hits act as seeds for initiating searches to find longer HSPs containing them. The word hits are extended in both directions along each sequence for as far as the cumulative alignment score can be increased. Cumulative scores are calculated using, e.g., for nucleotide sequences, the parameters M (reward score for a pair of matching residues; always > 0) and N (penalty score for mismatching residues; always < 0). For amino acid sequences, a scoring matrix is used to calculate the cumulative score. Extension of the word hits in each direction are halted when: the cumulative alignment score falls off by the quantity X from its maximum achieved value; the cumulative score goes to zero or below, due to the accumulation of one or more negative-scoring residue alignments; or the end of either sequence is reached. The BLAST algorithm parameters W , T , and X determine the sensitivity and speed of the alignment. The BLASTN program (for nucleotide sequences) uses as defaults a wordlength (W) of 11, an expectation (E) of 10, $M=5$, $N=-4$ and a comparison of both strands. For amino acid sequences, the BLASTP program uses as defaults a word length of 3, and expectation (E) of 10, and the BLOSUM62 scoring matrix (see Henikoff and Henikoff (1989) Proc. Nat'l. Acad. Sci. USA 89:10915-919) alignments (B) of 50, expectation (E) of 10, $M=5$, $N=-4$, and a comparison of both strands.

The BLAST algorithm also performs a statistical analysis of the similarity between two sequences (see, e.g., Karlin and Altschul (1993) Proc. Nat'l. Acad. Sci. USA 90:5873-5887). One measure of similarity provided by the BLAST algorithm is the smallest sum probability ($P(N)$), which provides an indication of the probability by which a match between two nucleotide or amino acid sequences would occur by chance. For example, a nucleic acid is considered similar to a reference sequence if the smallest sum probability in a comparison of the test nucleic acid to the reference nucleic acid is less than about 0.2, more preferably less than about 0.01, and most preferably less than about 0.001. Log values may be large negative numbers, e.g., 5, 10, 20, 30, 40, 70, 90, 110, 150, 170, etc.

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An indication that two nucleic acid sequences or polypeptides are substantially identical is that the polypeptide encoded by the first nucleic acid is immunologically cross reactive with the antibodies raised against the polypeptide encoded by the second nucleic acid, as described below. Thus, a polypeptide is typically substantially identical to a second polypeptide, e.g., where the two peptides differ only by conservative substitutions. Another indication that two nucleic acid sequences are substantially identical is that the two molecules or their complements hybridize to each other under stringent conditions, as described below. Yet another indication that two nucleic acid sequences are substantially identical is that the same primers can be used to amplify the sequences.

A "host cell" is a naturally occurring cell or a transformed cell that contains an expression vector and supports the replication or expression of the expression vector. Host cells may be cultured cells, explants, cells in vivo, and the like. Host cells may be prokaryotic cells such as *E. coli*, or eukaryotic cells such as yeast, insect, amphibian, or mammalian cells, such as CHO, HeLa, and the like (see, e.g., the American Type Culture Collection catalog or web site, www.atcc.org).

The terms "isolated," "purified," or "biologically pure" refer to material that is substantially or essentially free from components that normally accompany it as found in its native state. Purity and homogeneity are typically determined using analytical chemistry techniques such as polyacrylamide gel electrophoresis or high performance liquid chromatography. A protein or nucleic acid that is the predominant species present in a preparation is substantially purified. In particular, an isolated nucleic acid is separated from some open reading frames that naturally flank the gene and encode proteins other than protein encoded by the gene. The term "purified" in some embodiments denotes that a nucleic acid or protein gives rise to essentially one band in an electrophoretic gel. Preferably, it means that the nucleic acid or protein is at least 85% pure, more preferably at least 95% pure, and most preferably at least 99% pure. "Purify" or "purification" in other embodiments means removing at least one contaminant from the composition to be purified. In this sense, purification does not require that the purified compound be homogenous, e.g., 100% pure.

The terms "polypeptide," "peptide" and "protein" are used interchangeably herein to refer to a polymer of amino acid residues. The terms apply to amino acid polymers in which one or more amino acid residue is an artificial chemical mimetic of a corresponding naturally occurring amino acid, as well as to naturally occurring amino acid polymers, those containing modified residues, and non-naturally occurring amino acid polymers.

The term "amino acid" refers to naturally occurring and synthetic amino acids, as well as amino acid analogs and amino acid mimetics that function similarly to the naturally occurring amino acids. Naturally occurring amino acids are those encoded by the genetic code, as well as those amino acids that are later modified, e.g., hydroxyproline, γ -carboxyglutamate, and O-phosphoserine. Amino acid analogs refers to compounds that have the same basic chemical structure as a naturally occurring amino acid, e.g., an α carbon that is bound to a hydrogen, a carboxyl group, an amino group, and an R group, e.g., homoserine, norleucine, methionine sulfoxide, methionine methyl sulfonium. Such analogs may have modified R groups (e.g., norleucine) or modified peptide backbones, but retain the same basic chemical structure as a naturally occurring amino acid. Amino acid mimetics refers to chemical compounds that have a structure that is different from the general chemical structure of an amino acid, but that functions similarly to a naturally occurring amino acid.

Amino acids may be referred to herein by either their commonly known three letter symbols or by the one-letter symbols recommended by the IUPAC-IUB Biochemical Nomenclature Commission. Nucleotides, likewise, may be referred to by their commonly accepted single-letter codes.

"Conservatively modified variants" applies to both amino acid and nucleic acid sequences. With respect to particular nucleic acid sequences, conservatively modified variants refers to those nucleic acids which encode identical or essentially identical amino acid sequences, or where the nucleic acid does not encode an amino acid sequence, to essentially identical or associated, e.g., naturally contiguous, sequences. Because of the degeneracy of the genetic code, a large number of functionally identical nucleic acids encode most proteins. For instance, the codons GCA, GCC, GCG, and GCU all encode the amino acid alanine. Thus, at every position where an alanine is specified by a codon, the codon can be altered to another of the corresponding codons described without altering the encoded polypeptide. Such nucleic acid variations are "silent variations," which are one species of conservatively modified variations. Every nucleic acid sequence herein which encodes a polypeptide also describes silent variations of the nucleic acid. In certain contexts each codon in a nucleic acid (except AUG, which is ordinarily the only codon for methionine, and TGG, which is ordinarily the only codon for tryptophan) can be modified to yield a functionally identical molecule. Accordingly, a silent variation of a nucleic acid which encodes a polypeptide is implicit in a described sequence with respect to the expression product, but not necessarily with respect to actual probe sequences.

As to amino acid sequences, one of skill will recognize that individual substitutions, deletions, or additions to a nucleic acid, peptide, polypeptide, or protein sequence which alters, adds, or deletes a single amino acid or a small percentage of amino acids in the encoded sequence is a "conservatively modified variant" where the alteration results in the substitution of an amino acid with a chemically similar amino acid. Conservative substitution tables providing functionally similar amino acids are well known in the art. Such conservatively modified variants are in addition to and do not exclude polymorphic variants, interspecies homologs, and alleles of the invention. Typically conservative substitutions for one another: 1) Alanine (A), Glycine (G); 2) Aspartic acid (D), Glutamic acid (E); 3) Asparagine (N), Glutamine (Q); 4) Arginine (R), Lysine (K); 5) Isoleucine (I), Leucine (L), Methionine (M), Valine (V); 6) Phenylalanine (F), Tyrosine (Y), Tryptophan (W); 7) Serine (S), Threonine (T); and 8) Cysteine (C), Methionine (M) (see, e.g., Creighton (1984) Proteins Freeman).

Macromolecular structures such as polypeptide structures can be described in terms of various levels of organization. For a general discussion of this organization, see, e.g., Alberts, et al. (2001) Molecular Biology of the Cell (4th ed.) Garland Pub.; and Cantor and Schimmel (1980) Biophysical Chemistry Part I: The Conformation of Biological Macromolecules Freeman. "Primary structure" refers to the amino acid sequence of a particular peptide. "Secondary structure" refers to locally ordered, three dimensional structures within a polypeptide. These structures are commonly known as domains.

Domains are portions of a polypeptide that often form a compact unit of the polypeptide and are typically 25 to approximately 500 amino acids long. Typical domains are made up of sections of lesser organization such as stretches of β -sheet and α -helices. "Tertiary structure" refers to the complete three dimensional structure of a polypeptide monomer. "Quaternary structure" refers to the three dimensional structure formed, usually by the non-covalent association of independent tertiary units. Anisotropic terms are also known as energy terms.

"Nucleic acid" or "oligonucleotide" or "polynucleotide" or grammatical equivalents used herein means at least two nucleotides covalently linked together. Oligonucleotides are typically from about 5, 6, 7, 8, 9, 10, 12, 15, 25, 30, 40, 50, or more nucleotides in length, up to about 100 nucleotides in length. Nucleic acids and polynucleotides are a polymers of any length, including longer lengths, e.g., 200, 300, 500, 1000, 2000, 3000, 5000, 7000, 10,000, etc. A nucleic acid of the present invention will generally contain phosphodiester bonds,

although in some cases, nucleic acid analogs are included that may have at least one different linkage, e.g., phosphoramidate, phosphorothioate, phosphorodithioate, or O-methylphosphoramidite linkages (see Eckstein (1992) Oligonucleotides and Analogues: A Practical Approach Oxford University Press); and peptide nucleic acid backbones and linkages. Other analog nucleic acids include those with positive backbones; non-ionic backbones, and non-ribose backbones, including those described in U.S. Patent Nos. 5,235,033 and 5,034,506, and Chapters 6 and 7 of Sanghvi and Cook (eds. 1994) Carbohydrate Modifications in Antisense Research ASC Symposium Series 580. Nucleic acids containing one or more carbocyclic sugars are also included within one definition of nucleic acids. Modifications of the ribose-phosphate backbone may be done for a variety of reasons, e.g., to increase the stability and half-life of such molecules in physiological environments or as probes on a biochip. Mixtures of naturally occurring nucleic acids and analogs can be made, alternatively, mixtures of different nucleic acid analogs, and mixtures of naturally occurring nucleic acids and analogs may be made.

A variety of references disclose such nucleic acid analogs, including, e.g., phosphoramidate (Beaucage, et al. (1993) Tetrahedron 49:1925-1963 and references therein; Letsinger (1970) J. Org. Chem. 35:3800-3803; Sprinzl, et al. (1977) Eur. J. Biochem. 81:579-589; Letsinger, et al. (1986) Nucl. Acids Res. 14:3487-499; Sawai, et al. (1984) Chem. Lett. 805; Letsinger, et al. (1988) J. Am. Chem. Soc. 110:4470-4471; and Pauwels, et al. (1986), Chemica Scripta 26:141-149), phosphorothioate (Mag, et al. (1991) Nucl. Acids Res. 19:1437-441; and U.S. Patent No. 5,644,048), phosphorodithioate (Brill, et al. (1989) J. Am. Chem. Soc. 111:2321-2322), O-methylphosphoramidite linkages (see Eckstein (1992) Oligonucleotides and Analogues: A Practical Approach Oxford Univ. Press), and peptide nucleic acid backbones and linkages (see Egholm (1992) J. Am. Chem. Soc. 114:1895-897; Meier, et al. (1992) Angew. Chem. Int. Ed. Engl. 31:1008-1010; Nielsen (1993) Nature, 365:566-568; Carlsson, et al. (1996) Nature 380:207, each of which is incorporated by reference). Other analog nucleic acids include those with positive backbones (Denpoy, et al. (1995) Proc. Natl. Acad. Sci. USA 92:6097-101; non-ionic backbones (U.S. Patent Nos. 5,386,023, 5,637,684, 5,602,240, 5,216,141 and 4,469,863; Kiedrowski, et al. (1991) Angew. Chem. Int. Ed. Engl. 30:423-426; Letsinger, et al. (1988) J. Am. Chem. Soc. 110:4470-4471; Jung, et al. (1994) Nucleoside and Nucleotide 13:1597; Chapters 2 and 3, in Sanghvi and Cook (eds. 1994) Carbohydrate Modifications in Antisense Research ASC Symposium Series 580; Mesmaeker, et al. (1994) Bioorganic and Medicinal Chem. Lett. 4:395-398; Jeffs,

et al. (1994) J. Biomolecular NMR 34:17-xx; Horn, et al. (1996) Tetrahedron Lett. 37:743-xxx) and non-ribose backbones, including those described in U.S. Patent Nos. 5,235,033 and 5,034,506, and Chapters 6 and 7, in Sanghvi and Cook (eds. 1994) Carbohydrate Modifications in Antisense Research ASC Symposium Series 580. Nucleic acids containing one or more carbo-cyclic sugars are also included within one definition of nucleic acids (see Jenkins, et al. (1995) Chem. Soc. Rev. pp 169-176). Several nucleic acid analogs are described in Rawls (p. 35 June 2, 1997) C&E News. Each of these references is hereby expressly incorporated by reference.

Particularly preferred are peptide nucleic acids (PNA) which includes peptide nucleic acid analogs. These backbones are substantially non-ionic under neutral conditions, in contrast to the highly charged phosphodiester backbone of naturally occurring nucleic acids. This results in two advantages. First, the PNA backbone exhibits improved hybridization kinetics. PNAs have larger changes in the melting temperature (T_m) for mismatched versus perfectly matched base pairs. DNA and RNA typically exhibit a 2-4° C drop in T_m for an internal mismatch. With the non-ionic PNA backbone, the drop is closer to 7-9° C. Similarly, due to their non-ionic nature, hybridization of the bases attached to these backbones is relatively insensitive to salt concentration. In addition, PNAs are not degraded by cellular enzymes, and thus can be more stable.

The nucleic acids may be single stranded or double stranded, as specified, or contain portions of both double stranded or single stranded sequence. As will be appreciated by those in the art, the depiction of a single strand also defines the sequence of the complementary strand; thus the sequences described herein also provide the complement of the sequence. The nucleic acid may be DNA, both genomic and cDNA, RNA, or a hybrid, where the nucleic acid may contain combinations of deoxyribo- and ribo-nucleotides, and combinations of bases, including uracil, adenine, thymine, cytosine, guanine, inosine, xanthine hypoxanthine, isocytosine, isoguanine, etc. "Transcript" typically refers to a naturally occurring RNA, e.g., a pre-mRNA, hnRNA, or mRNA. As used herein, the term "nucleoside" includes nucleotides and nucleoside and nucleotide analogs, and modified nucleosides such as amino modified nucleosides. In addition, "nucleoside" includes non-naturally occurring analog structures. Thus, e.g., the individual units of a peptide nucleic acid, each containing a base, are referred to herein as a nucleoside.

A "label" or a "detectable moiety" is a composition detectable by spectroscopic,

photochemical, biochemical, immunochemical, chemical, or other physical means. For example, useful labels include ^{32}P , fluorescent dyes, electron-dense reagents, enzymes (e.g., as commonly used in an ELISA), biotin, digoxigenin, or haptens and proteins or other entities which can be made detectable, e.g., by incorporating a radiolabel into the peptide or used to detect antibodies specifically reactive with the peptide. The labels may be incorporated into the ovarian cancer nucleic acids, proteins and antibodies at any position. Any method known in the art for conjugating the antibody to the label may be employed, including those methods described by Hunter, et al. (1962) Nature 144:945-xxx; David, et al. (1974) Biochemistry 13:1014-1021; Pain, et al. (1981) J. Immunol. Meth. 40:219-230; and Nygren (1982) J. Histochem. and Cytochem. 30:407-412.

An "effector" or "effector moiety" or "effector component" is a molecule that is bound (or linked, or conjugated), either covalently, through a linker or a chemical bond, or non-covalently, through ionic, van der Waals, electrostatic, or hydrogen bonds, to an antibody. The "effector" can be a variety of molecules including, e.g., detection moieties including radioactive compounds, fluorescent compounds, an enzyme or substrate, tags such as epitope tags, a toxin; activatable moieties, a chemotherapeutic agent; a lipase; an antibiotic; or a radioisotope emitting "hard" e.g., beta radiation.

A "labeled nucleic acid probe or oligonucleotide" is one that is bound, either covalently, through a linker or a chemical bond, or non-covalently, through ionic, van der Waals, electrostatic, or hydrogen bonds to a label such that the presence of the probe may be detected by detecting the presence of the label bound to the probe. Alternatively, method using high affinity interactions may achieve the same results where one of a pair of binding partners binds to the other, e.g., biotin, streptavidin.

As used herein a "nucleic acid probe or oligonucleotide" is a nucleic acid capable of binding to a target nucleic acid of complementary sequence through one or more types of chemical bonds, usually through complementary base pairing, usually through hydrogen bond formation. As used herein, a probe may include natural (e.g., A, G, C, or T) or modified bases (7-deazaguanosine, inosine, etc.). In addition, the bases in a probe may be joined by a linkage other than a phosphodiester bond, so long as it does not functionally interfere with hybridization. Thus, e.g., probes may be peptide nucleic acids in which the constituent bases are joined by peptide bonds rather than phosphodiester linkages. Probes may bind target sequences lacking complete complementarity with the probe sequence depending upon the

stringency of the hybridization conditions. The probes are preferably directly labeled, e.g., with isotopes, chromophores, lumiphores, chromogens, or indirectly labeled such as with biotin to which a streptavidin complex may later bind. By assaying for the presence or absence of the probe, one can detect the presence or absence of the select sequence or subsequence. Diagnosis or prognosis may be based at the genomic level, or at the level of RNA or protein expression.

The term "recombinant" when used with reference, e.g., to a cell, or nucleic acid, protein, or vector, indicates that the cell, nucleic acid, protein or vector, has been modified by the introduction of a heterologous nucleic acid or protein or the alteration of a native nucleic acid or protein, or that the cell is derived from a cell so modified. Thus, e.g., recombinant cells express genes that are not found within the native (non-recombinant) form of the cell or express native genes that are otherwise abnormally expressed, under expressed or not expressed at all. By the term "recombinant nucleic acid" herein is meant nucleic acid, originally formed in vitro, in general, by the manipulation of nucleic acid, e.g., using polymerases and endonucleases, in a form not normally found in nature. In this manner, operably linkage of different sequences is achieved. Thus an isolated nucleic acid, in a linear form, or an expression vector formed in vitro by ligating DNA molecules that are not normally joined, are both considered recombinant for the purposes of this invention. It is understood that once a recombinant nucleic acid is made and reintroduced into a host cell or organism, it will replicate non-recombinantly, e.g., using the in vivo cellular machinery of the host cell rather than in vitro manipulations; however, such nucleic acids, once produced recombinantly, although subsequently replicated non-recombinantly, are still considered recombinant for the purposes of the invention. Similarly, a "recombinant protein" is a protein made using recombinant techniques, e.g., through the expression of a recombinant nucleic acid as depicted above.

The term "heterologous" when used with reference to portions of a nucleic acid indicates that the nucleic acid comprises two or more subsequences that are not normally found in the same relationship to each other in nature. For instance, the nucleic acid is typically recombinantly produced, having two or more sequences, e.g., from unrelated genes arranged to make a new functional nucleic acid, e.g., a promoter from one source and a coding region from another source. Similarly, a heterologous protein will often refer to two or more subsequences that are not found in the same relationship to each other in nature (e.g., a fusion protein).

A "promoter" is defined as an array of nucleic acid control sequences that direct transcription of a nucleic acid. As used herein, a promoter includes necessary nucleic acid sequences near the start site of transcription, such as, in the case of a polymerase II type promoter, a TATA element. A promoter also optionally includes distal enhancer or repressor elements, which can be located as much as several thousand base pairs from the start site of transcription. A "constitutive" promoter is a promoter that is active under most environmental and developmental conditions. An "inducible" promoter is a promoter that is active under environmental or developmental regulation. The term "operably linked" refers to a functional linkage between a nucleic acid expression control sequence (such as a promoter, or array of transcription factor binding sites) and a second nucleic acid sequence, e.g., wherein the expression control sequence directs transcription of the nucleic acid corresponding to the second sequence.

An "expression vector" is a nucleic acid construct, generated recombinantly or synthetically, with a series of specified nucleic acid elements that permit transcription of a particular nucleic acid in a host cell. The expression vector can be part of a plasmid, virus, or nucleic acid fragment. Typically, the expression vector includes a nucleic acid to be transcribed operably linked to a promoter.

The phrase "selectively (or specifically) hybridizes to" refers to the binding, duplexing, or hybridizing of a molecule only to a particular nucleotide sequence under stringent hybridization conditions when that sequence is present in a complex mixture (e.g., total cellular or library DNA or RNA).

The phrase "stringent hybridization conditions" refers to conditions under which a probe will hybridize to its target subsequence, typically in a complex mixture of nucleic acids, but to no other sequences. Stringent conditions are sequence-dependent and will be different in different circumstances. Longer sequences hybridize specifically at higher temperatures. An extensive guide to the hybridization of nucleic acids is found in "Overview of principles of hybridization and the strategy of nucleic acid assays" in Tijssen (1993)

Hybridization with Nucleic Probes (Laboratory Techniques in Biochemistry and Molecular Biology) (vol. 24) Elsevier. Generally, stringent conditions are selected to be about 5-10° C lower than the thermal melting point (T_m) for the specific sequence at a defined ionic strength pH. The T_m is the temperature (under defined ionic strength, pH, and nucleic concentration) at which 50% of the probes complementary to the target hybridize to the target

sequence at equilibrium (as the target sequences are present in excess, at T_m , 50% of the probes are occupied at equilibrium). Stringent conditions will be those in which the salt concentration is less than about 1.0 M sodium ion, typically about 0.01 to 1.0 M sodium ion concentration (or other salts) at pH 7.0 to 8.3 and the temperature is at least about 30° C for short probes (e.g., 10 to 50 nucleotides) and at least about 60° C for long probes (e.g., greater than 50 nucleotides). Stringent conditions may also be achieved with the addition of destabilizing agents such as formamide. For selective or specific hybridization, a positive signal is typically at least two times background, preferably 10 times background hybridization. Exemplary stringent hybridization conditions can be as following: 50% formamide, 5x SSC, and 1% SDS, incubating at 42° C, or, 5x SSC, 1% SDS, incubating at 65° C, with wash in 0.2x SSC, and 0.1% SDS at 65° C. For PCR, a temperature of about 36° C is typical for low stringency amplification, although annealing temperatures may vary between about 32-48° C depending on primer length. For high stringency PCR amplification, a temperature of about 62° C is typical, although high stringency annealing temperatures can range from about 50° C to about 65° C, depending on the primer length and specificity. Typical cycle conditions for both high and low stringency amplifications include a denaturation phase of 90-95° C for 30-120 sec, an annealing phase lasting 30-120 sec, and an extension phase of about 72° C for 1-2 min. Protocols and guidelines for low and high stringency amplification reactions are available, e.g., in Innis, et al. (1990) PCR Protocols: A Guide to Methods and Applications Academic Press, N.Y.

Nucleic acids that do not hybridize to each other under stringent conditions are still substantially identical if the polypeptides which they encode are substantially identical. This occurs, e.g., when a copy of a nucleic acid is created using the maximum codon degeneracy permitted by the genetic code. In such cases, the nucleic acids typically hybridize under moderately stringent hybridization conditions. Exemplary "moderately stringent hybridization conditions" include a hybridization in a buffer of 40% formamide, 1 M NaCl, 1% SDS at 37° C, and a wash in 1x SSC at 45° C. A positive hybridization is at least twice background. Alternative hybridization and wash conditions can be utilized to provide conditions of similar stringency. Additional guidelines for determining hybridization parameters are provided, e.g., Ausubel, et al. (ed. 1991 and supplements) Current Protocols in Molecular Biology Lippincott.

The phrase "functional effects" in the context of assays for testing compounds that

modulate activity of an ovarian cancer protein includes the determination of a parameter that is indirectly or directly under the influence of the ovarian cancer protein or nucleic acid, e.g., a functional, physical, physiological, or chemical effect, such as the ability to decrease ovarian cancer. It includes ligand binding activity; cell growth on soft agar; anchorage dependence; contact inhibition and density limitation of growth; cellular proliferation; cellular transformation; growth factor or serum dependence; tumor specific marker levels; invasiveness into Matrigel; tumor growth and metastasis in vivo; mRNA and protein expression in cells undergoing metastasis, and other characteristics of ovarian cancer cells. "Functional effects" include in vitro, in vivo, and ex vivo activities.

By "determining the functional effect" is meant assaying for a compound that increases or decreases a parameter that is indirectly or directly under the influence of an ovarian cancer protein sequence, e.g., functional, enzymatic, physical, physiological, and chemical effects. Such functional effects can be measured by any means known to those skilled in the art, e.g., changes in spectroscopic characteristics (e.g., fluorescence, absorbance, refractive index), hydrodynamic (e.g., shape), chromatographic, or solubility properties for the protein, measuring inducible markers or transcriptional activation of the ovarian cancer protein; measuring binding activity or binding assays, e.g., binding to antibodies or other ligands, and measuring cellular proliferation. Determination of the functional effect of a compound on ovarian cancer can also be performed using ovarian cancer assays known to those of skill in the art such as in vitro assays, e.g., cell growth on soft agar; anchorage dependence; contact inhibition and density limitation of growth; cellular proliferation; cellular transformation; growth factor or serum dependence; tumor specific marker levels; invasiveness into Matrigel; tumor growth and metastasis in vivo; mRNA and protein expression in cells undergoing metastasis, and other characteristics of ovarian cancer cells. The functional effects can be evaluated by means known to those skilled in the art, e.g., microscopy for quantitative or qualitative measures of alterations in morphological features, measurement of changes in RNA or protein levels for ovarian cancer-associated sequences, measurement of RNA stability, or identification of downstream or reporter gene expression (CAT, luciferase, β -gal, GFP, and the like), e.g., via chemiluminescence, fluorescence, colorimetric reactions, antibody binding, inducible markers, and ligand binding assays.

"Inhibitors", "activators", and "modulators" of ovarian cancer polynucleotide and polypeptide sequences are used to refer to activating, inhibitory, or modulating molecules or compounds identified using in vitro and in vivo assays of ovarian cancer polynucleotide and

polypeptide sequences. Inhibitors are compounds that, e.g., bind to, partially or totally block activity, decrease, prevent, delay activation, inactivate, desensitize, or down regulate the activity or expression of ovarian cancer proteins, e.g., antagonists. Antisense or inhibitory nucleic acids may inhibit expression and subsequent function of the protein. "Activators" are compounds that increase, open, activate, facilitate, enhance activation, sensitize, agonize, or up regulate ovarian cancer protein activity. Inhibitors, activators, or modulators also include genetically modified versions of ovarian cancer proteins, e.g., versions with altered activity, as well as naturally occurring and synthetic ligands, antagonists, agonists, antibodies, small chemical molecules, and the like. Assays for inhibitors and activators include, e.g., expressing the ovarian cancer protein in vitro, in cells, or cell membranes, applying putative modulator compounds, and then determining the functional effects on activity, as described above. Activators and inhibitors of ovarian cancer can also be identified by incubating ovarian cancer cells with the test compound and determining increases or decreases in the expression of one or more ovarian cancer proteins, e.g., 1, 2, 3, 4, 5, 10, 15, 20, 25, 30, 40, 50, or more ovarian cancer proteins, such as ovarian cancer proteins encoded by the sequences set out in Tables 1-26.

Samples or assays comprising ovarian cancer proteins that are treated with a potential activator, inhibitor, or modulator are compared to control samples without the inhibitor, activator, or modulator to examine the extent of inhibition. Control samples (untreated with inhibitors) are assigned a relative protein activity value of 100%. Inhibition of a polypeptide is achieved when the activity value relative to the control is about 80%, preferably 50%, more preferably 25% or less. Activation of an ovarian cancer polypeptide is achieved when the activity value relative to the control (untreated with activators) is 110%, more preferably 150%, more preferably 200-500% (e.g., 2-5 fold higher relative to the control), more preferably 1000-3000% higher.

The phrase "changes in cell growth" refers to a change in cell growth and proliferation characteristics in vitro or in vivo, e.g., cell viability, formation of foci, anchorage independence, semi-solid or soft agar growth, change in contact inhibition or density limitation of growth, loss of growth factor or serum requirements, change in cell morphology, gain or loss of immortalization, gain or loss of tumor specific markers, ability to form or suppress tumors when injected into suitable animal hosts, and/or immortalization of the cell. See, e.g., pp. 231-241 in Freshney (1994) Culture of Animal Cells: A Manual of Basic Technique (3d ed.) Wiley-Liss.

"Tumor cell" refers to pre-cancerous, cancerous, and normal cells in a tumor.

"Cancer cells," "transformed" cells or "transformation" in tissue culture, refers to spontaneous or induced phenotypic changes that do not necessarily involve the uptake of new genetic material. Although transformation can arise from infection with a transforming virus and incorporation of new genomic DNA, or uptake of exogenous DNA, it can also arise spontaneously or following exposure to a carcinogen, thereby mutating an endogenous gene. Transformation is typically associated with phenotypic changes, such as immortalization of cells, aberrant growth control, non-morphological changes, and/or malignancy. See, Freshney (1994) Culture of Animal Cells.

"Antibody" refers to a polypeptide comprising a framework region from an immunoglobulin gene or fragments thereof that specifically binds and recognizes an antigen. The recognized immunoglobulin genes include the kappa, lambda, alpha, gamma, delta, epsilon, and mu constant region genes, as well as the myriad immunoglobulin variable region genes. Light chains are classified as either kappa or lambda. Heavy chains are classified as gamma, mu, alpha, delta, or epsilon, which in turn define the immunoglobulin classes, IgG, IgM, IgA, IgD, and IgE, respectively. Typically, the antigen-binding region of an antibody or its functional equivalent will be most critical in specificity and affinity of binding. See, e.g., Paul (ed. 1999) Fundamental Immunology (4th ed.) Raven.

An exemplary immunoglobulin (antibody) structural unit comprises a tetramer. Each tetramer is composed of two identical pairs of polypeptide chains, each pair having one "light" (about 25 kD) and one "heavy" chain (about 50-70 kD). The N-terminus of each chain defines a variable region of about 100 to 110 or more amino acids primarily responsible for antigen recognition. The terms variable light chain (V_L) and variable heavy chain (V_H) refer to these light and heavy chains respectively.

Antibodies exist, e.g., as intact immunoglobulins or as a number of well-characterized fragments produced by digestion with various peptidases. Thus, e.g., pepsin digests an antibody below the disulfide linkages in the hinge region to produce $F(ab')_2$, a dimer of Fab which itself is a light chain joined to V_H-C_H1 by a disulfide bond. The $F(ab')_2$ may be reduced under mild conditions to break the disulfide linkage in the hinge region, thereby converting the $F(ab')_2$ dimer into an Fab' monomer. The Fab' monomer is essentially Fab with part of the hinge region. See Paul (ed. 1999) Fundamental Immunology (4th ed.) Raven. While various antibody fragments are defined in terms of the digestion of an intact antibody,

one of skill will appreciate that such fragments may be synthesized de novo either chemically or by using recombinant DNA methodology. Thus, the term antibody, as used herein, also includes antibody fragments either produced by the modification of whole antibodies, or those synthesized de novo using recombinant DNA methodologies (e.g., single chain Fv) or those identified using phage display libraries. See, e.g., McCafferty, et al. (1990) Nature 348:552-554.

For preparation of antibodies, e.g., recombinant, monoclonal, or polyclonal antibodies, many techniques known in the art can be used (see, e.g., Kohler and Milstein (1975) Nature 256:495-497; Kozbor, et al. (1983) Immunology Today 4:72; Cole, et al., pp. 77-96 in Reisfeld and Sell (1985) Monoclonal Antibodies and Cancer Therapy Liss; Coligan (1991) Current Protocols in Immunology Lippincott; Harlow and Lane (1988) Antibodies: A Laboratory Manual CSH Press; and Goding (1986) Monoclonal Antibodies: Principles and Practice (2d ed.) Academic Press. Techniques for the production of single chain antibodies (U.S. Patent 4,946,778) can be adapted to produce antibodies to polypeptides of this invention. Transgenic mice, or other organisms, e.g., other mammals, may be used to express humanized antibodies. Alternatively, phage display technology can be used to identify antibodies and heteromeric Fab fragments that specifically bind to selected antigens. See, e.g., McCafferty, et al. (1990) Nature 348:552-554; and Marks, et al. (1992) Biotechnology 10:779-783.

A "chimeric antibody" is an antibody molecule in which (a) the constant region, or a portion thereof, is altered, replaced or exchanged so that the antigen binding site (variable region) is linked to a constant region of a different or altered class, effector function and/or species, or an entirely different molecule which confers new properties to the chimeric antibody, e.g., an enzyme, toxin, hormone, growth factor, drug, etc.; or (b) the variable region, or a portion thereof, is altered, replaced or exchanged with a variable region having a different or altered antigen specificity.

Identification of ovarian cancer-associated sequences

In one aspect, the expression levels of genes are determined in different patient samples for which diagnosis information is desired, to provide expression profiles. An expression profile of a particular sample is essentially a "fingerprint" of the state of the sample, while two states may have any particular gene similarly expressed, the evaluation of a number of genes simultaneously allows the generation of a gene expression profile that is

characteristic of the state of the cell. That is, normal tissue (e.g., normal ovarian or other tissue) may be distinguished from cancerous or metastatic cancerous tissue of the ovarian, or ovarian cancer tissue or metastatic ovarian cancerous tissue can be compared with tissue samples of ovarian and other tissues from surviving cancer patients. By comparing expression profiles of tissue in known different ovarian cancer states, information regarding which genes are important (including both up- and down-regulation of genes) in each of these states is obtained. Molecular profiling may distinguish subtypes of a currently collective disease designation, e.g., different forms of a cancer.

The identification of sequences that are differentially expressed in ovarian cancer versus non-ovarian cancer tissue allows the use of this information in a number of ways. For example, a particular treatment regime may be evaluated: does a chemotherapeutic drug act to down-regulate ovarian cancer, and thus tumor growth or recurrence, in a particular patient. Alternatively, does existing treatment induce expression of a target. Similarly, diagnosis and treatment outcomes may be done or confirmed by comparing patient samples with the known expression profiles. Metastatic tissue can also be analyzed to determine the stage of ovarian cancer in the tissue or origin of the primary tumor. Furthermore, these gene expression profiles (or individual genes) allow screening of drug candidates with an eye to mimicking or altering a particular expression profile; e.g., screening can be done for drugs that suppress the ovarian cancer expression profile. This may be done by making biochips comprising sets of the important ovarian cancer genes, which can then be used in these screens. These methods can also be based on evaluating protein expression; that is, protein expression levels of the ovarian cancer proteins can be evaluated for diagnostic purposes or to screen candidate agents. In addition, the ovarian cancer nucleic acid sequences can be administered for gene therapy purposes, including the administration of antisense or RNAi nucleic acids, or the ovarian cancer proteins (including antibodies and other modulators thereof) administered as therapeutic drugs.

Thus the present invention provides nucleic acid and protein sequences that are differentially expressed in ovarian cancer relative to normal tissues and/or non-malignant tissues, herein termed "ovarian cancer sequences." As outlined below, ovarian cancer sequences include those that are up-regulated (e.g., expressed at a higher level) in ovarian cancer, as well as those that are down-regulated (e.g., expressed at a lower level). In a preferred embodiment, the ovarian cancer sequences are from humans; however, as will be appreciated by those in the art, ovarian cancer sequences from other organisms may be useful

in animal models of disease and drug evaluation; thus, other ovarian cancer sequences are provided, from vertebrates, including mammals, including rodents (rats, mice, hamsters, guinea pigs, etc.), primates, farm animals (including sheep, goats, pigs, cows, horses, etc.) and pets (e.g., dogs, cats, etc.). Ovarian cancer sequences, e.g., counterpart genes, from other organisms may be obtained using the techniques outlined below.

Ovarian cancer sequences can include both nucleic acid and amino acid sequences. Ovarian cancer nucleic acid sequences are useful in a variety of applications, including diagnostic applications, which will detect naturally occurring nucleic acids. Screening applications; e.g., biochips comprising nucleic acid probes or PCR microtiter plates with selected probes to the ovarian cancer sequences, are also provided.

An ovarian cancer sequence can be initially identified by substantial nucleic acid and/or amino acid sequence homology to the ovarian cancer sequences outlined herein. Such homology can be based upon the overall nucleic acid or amino acid sequence, and is generally determined as outlined below, using either homology programs or hybridization conditions.

For identifying ovarian cancer-associated sequences, the ovarian cancer screen typically includes comparing genes identified in different tissues, e.g., normal and cancerous tissues, or tumor tissue samples from patients who have metastatic disease vs. non metastatic tissue. Other suitable tissue comparisons include comparing ovarian cancer samples with metastatic cancer samples from other cancers, such as lung, ovarian, gastrointestinal cancers, etc. Samples of different stages of ovarian cancer, e.g., survivor tissue, drug resistant states, and tissue undergoing metastasis, are applied to biochips comprising nucleic acid probes. The samples are first microdissected, if applicable, and treated for the preparation of mRNA. Suitable biochips are commercially available, e.g., from Affymetrix. Gene expression profiles as described herein are generated and the data analyzed.

In one embodiment, the genes showing changes in expression as between normal and disease states are compared to genes expressed in other normal tissues, preferably normal ovarian, but also including, and not limited to, lung, heart, brain, liver, ovarian, kidney, muscle, colon, small intestine, large intestine, spleen, bone, and/or placenta. In a preferred embodiment, those genes identified during the ovarian cancer screen that are expressed in any significant amount in other tissues are removed from the profile, although in some embodiments, expression in non-essential tissues may be tolerated. That is, when screening for drugs, it is usually preferable that the target be disease specific, to minimize possible side

effects by interaction with target present in other organs.

In a preferred embodiment, ovarian cancer sequences are those that are up-regulated in ovarian cancer; that is, the expression of these genes is higher in the ovarian cancer tissue as compared to non-cancerous tissue. "Up-regulation" as used herein often means at least about a two-fold change, preferably at least about a three fold change, with at least about five-fold or higher being preferred. Other embodiments are directed to sequences up regulated in non-malignant conditions relative to normal.

Unigene cluster identification numbers and accession numbers herein refer to the GenBank sequence database and the sequences of the accession numbers are hereby expressly incorporated by reference. GenBank is known in the art, see, e.g., Benson, et al. (1998) Nucl. Acids Res. 26:1-7; and <http://www.ncbi.nlm.nih.gov/>. Sequences are also available in other databases, e.g., European Molecular Biology Laboratory (EMBL) and DNA Database of Japan (DDBJ). In some situations, the sequences may be derived from assembly of available sequences or be predicted from genomic DNA using exon prediction algorithms, e.g., FGENESH. See Salamov and Solovyev (2000) Genome Res. 10:516-522. In other situations, sequences have been derived from cloning and sequencing of isolated nucleic acids.

In another preferred embodiment, ovarian cancer sequences are those that are down-regulated in ovarian cancer; that is, the expression of these genes is lower in ovarian cancer tissue as compared to non-cancerous tissue. "Down-regulation" as used herein often means at least about a two-fold change, preferably at least about a three-fold change, with at least about five-fold or higher being preferred.

Informatics

The ability to identify genes that are over or under expressed in ovarian cancer can additionally provide high-resolution, high-sensitivity datasets which can be used in the areas of diagnostics, therapeutics, drug development, pharmacogenetics, protein structure, biosensor development, and other related areas. Expression profiles can be used in diagnostic or prognostic evaluation of patients with ovarian cancer. Subcellular toxicological information can be generated to better direct drug structure and activity correlation (see Anderson (June 11-12, 1998) Pharmaceutical Proteomics: Targets, Mechanism, and Function, paper presented at the IBC Proteomics conference, Coronado, CA) or in a biological sensor device to predict the likely toxicological effect of chemical exposures and likely tolerable

exposure thresholds (see U.S. Patent No. 5,811,231). Similar advantages accrue from datasets relevant to other biomolecules and bioactive agents (e.g., nucleic acids, saccharides, lipids, drugs, and the like).

Thus, in another embodiment, the present invention provides a database that includes at least one set of assay data. The data contained in the database is acquired, e.g., using array analysis either singly or in a library format. The database can be in a form in which data can be maintained and transmitted, but is preferably an electronic database, and can be maintained on any electronic device allowing for the storage of and access to the database, such as a personal computer, but is preferably distributed on a wide area network, such as the World Wide Web.

The focus of the present section on databases that include peptide sequence data is for clarity of illustration only. It will be apparent to those of skill in the art that similar databases can be assembled for any assay data acquired using an assay of the invention.

The compositions and methods for identifying and/or quantitating the relative and/or absolute abundance of a variety of molecular and macromolecular species from a biological sample undergoing ovarian cancer, e.g., the identification of ovarian cancer-associated sequences described herein, provide an abundance of information which can be correlated with pathological conditions, predisposition to disease, drug testing, therapeutic monitoring, gene-disease causal linkages, identification of correlates of immunity and physiological status, and outcome data, among others. Although data generated from the assays of the invention is suited for manual review and analysis, in a preferred embodiment, data processing using high-speed computers is utilized.

An array of methods for indexing and retrieving biomolecular information is known in the art. For example, U.S. Patents 6,023,659 and 5,966,712 disclose a relational database system for storing biomolecular sequence information in a manner that allows sequences to be catalogued and searched according to one or more protein function hierarchies. U.S. Patent 5,953,727 discloses a relational database having sequence records containing information in a format that allows a collection of partial-length DNA sequences to be catalogued and searched according to association with one or more sequencing projects for obtaining full-length sequences from the collection of partial length sequences. U.S. Patent 5,706,498 discloses a gene database retrieval system for making a retrieval of a gene sequence similar to a sequence data item in a gene database based on the degree of similarity between a key sequence and a target sequence. U.S. Patent 5,538,897 discloses a method

using mass spectroscopy fragmentation patterns of peptides to identify amino acid sequences in computer databases by comparison of predicted mass spectra with experimentally-derived mass spectra using a closeness-of-fit measure. U.S. Patent 5,926,818 discloses a multi-dimensional database comprising a functionality for multi-dimensional data analysis described as on-line analytical processing (OLAP), which entails the consolidation of projected and actual data according to more than one consolidation path or dimension. U.S. Patent 5,295,261 reports a hybrid database structure in which the fields of each database record are divided into two classes, navigational and informational data, with navigational fields stored in a hierarchical topological map which can be viewed as a tree structure or as the merger of two or more such tree structures.

Fundamentals of bioinformatics are provided, e.g., in Mount, et al. (2001) Bioinformatics: Sequence and Genome Analysis CSH Press, NY; Durbin, et al. (eds. 1999) Biological Sequence Analysis: Probabilistic Models of Proteins and Nucleic Acids Cambridge Univ. Press; Baxevanis and Ouellette (eds. 1998) Bioinformatics: A Practical Guide to the Analysis of Genes and Proteins (2d ed.) Wiley-Liss; Rashidi and Buehler (1999) Bioinformatics: Basic Applications in Biological Science and Medicine CRC Press; Setubal, et al. (eds 1997) Introduction to Computational Molecular Biology Brooks/Cole; Misener and Kravetz (eds. 2000) Bioinformatics: Methods and Protocols Humana Press; Higgins and Taylor (eds. 2000) Bioinformatics: Sequence, Structure, and Databanks: A Practical Approach Oxford Univ. Press; Brown (2001) Bioinformatics: A Biologist's Guide to Biocomputing and the Internet Eaton Pub.; Han and Kamber (2000) Data Mining: Concepts and Techniques Kaufmann Pub.; and Waterman (1995) Introduction to Computational Biology: Maps, Sequences, and Genomes Chap and Hall.

The present invention provides a computer database comprising a computer and software for storing in computer-retrievable form assay data records cross-tabulated, e.g., with data specifying the source of the target-containing sample from which each sequence specificity record was obtained.

In an exemplary embodiment, at least one of the sources of target-containing sample is from a control tissue sample known to be free of pathological disorders. In a variation, at least one of the sources is a known pathological tissue specimen, e.g., a neoplastic lesion or another tissue specimen to be analyzed for ovarian cancer. In another variation, assay records cross-tabulate one or more of the following parameters for a target species in a sample: (1) a unique identification code, which can include, e.g., a target molecular structure and/or

characteristic separation coordinate (e.g., electrophoretic or genomic position coordinates); (2) sample source; and (3) absolute and/or relative quantity of target species present in the sample.

The invention also provides for the storage and retrieval of a collection of target data in a computer data storage apparatus, which can include magnetic disks, optical disks, magneto-optical disks, DRAM, SRAM, SGRAM, SDRAM, RDRAM, DDR RAM, magnetic bubble memory devices, and other data storage devices, including CPU registers and on-CPU data storage arrays. Typically, the target data records are stored as a bit pattern in an array of magnetic domains on a magnetizable medium or as an array of charge states or transistor gate states, such as an array of cells in a DRAM device (e.g., each cell comprised of a transistor and a charge storage area, which may be on the transistor). In one embodiment, the invention provides such storage devices, and computer systems built therewith, comprising a bit pattern encoding a protein expression fingerprint record comprising unique identifiers for at least 10 target data records cross-tabulated with target source.

When the target is a peptide or nucleic acid, the invention preferably provides a method for identifying related peptide or nucleic acid sequences, comprising performing a computerized comparison between a peptide or nucleic acid sequence assay record stored in or retrieved from a computer storage device or database and at least one other sequence. The comparison can include a sequence analysis or comparison algorithm or computer program embodiment thereof (e.g., FASTA, TFASTA, GAP, BESTFIT) and/or the comparison may be of the relative amount of a peptide or nucleic acid sequence in a pool of sequences determined from a polypeptide or nucleic acid sample of a specimen.

The invention also preferably provides a magnetic disk, such as an IBM-compatible (DOS, Windows, Windows95/98/2000, Windows NT, OS/2) or other format (e.g., Linux, SunOS, Solaris, AIX, SCO Unix, VMS, MV, Macintosh, etc.) floppy diskette or hard (fixed, Winchester) disk drive, comprising a bit pattern encoding data from an assay of the invention in a file format suitable for retrieval and processing in a computerized sequence analysis, comparison, or relative quantitation method.

The invention also provides a network, comprising a plurality of computing devices linked via a data link, such as an Ethernet cable (coax or 10BaseT), telephone line, ISDN line, wireless network, optical fiber, or other suitable signal transmission medium, whereby at least one network device (e.g., computer, disk array, etc.) comprises a pattern of magnetic domains (e.g., magnetic disk) and/or charge domains (e.g., an array of DRAM cells)

composing a bit pattern encoding data acquired from an assay of the invention.

The invention also provides a method for transmitting assay data that includes generating an electronic signal on an electronic communications device, such as a modem, ISDN terminal adapter, DSL, cable modem, ATM switch, or the like, wherein the signal includes (in native or encrypted format) a bit pattern encoding data from an assay or a database comprising a plurality of assay results obtained by the method of the invention.

In a preferred embodiment, the invention provides a computer system for comparing a query target to a database containing an array of data structures, such as an assay result obtained by the method of the invention, and ranking database targets based on the degree of identity and gap weight to the target data. A central processor is preferably initialized to load and execute the computer program for alignment and/or comparison of the assay results. Data for a query target is entered into the central processor via an I/O device. Execution of the computer program results in the central processor retrieving the assay data from the data file, which comprises a binary description of an assay result.

The target data or record and the computer program can be transferred to secondary memory, which is typically random access memory (e.g., DRAM, SRAM, SGRAM, or SDRAM). Targets are ranked according to the degree of correspondence between a selected assay characteristic (e.g., binding to a selected affinity moiety) and the same characteristic of the query target and results are output via an I/O device. For example, a central processor can be a conventional computer (e.g., Intel Pentium, PowerPC, Alpha, PA-8000, SPARC, MIPS 4400, MIPS 10000, VAX, etc.); a program can be a commercial or public domain molecular biology software package (e.g., UWGCG Sequence Analysis Software, Darwin); a data file can be an optical or magnetic disk, a data server, a memory device (e.g., DRAM, SRAM, SGRAM, SDRAM, EPROM, bubble memory, flash memory, etc.); an I/O device can be a terminal comprising a video display and a keyboard, a modem, an ISDN terminal adapter, an Ethernet port, a punched card reader, a magnetic strip reader, or other suitable I/O device.

The invention also preferably provides the use of a computer system, e.g., which typically comprises one or more of: (1) a computer; (2) a stored bit pattern encoding a collection of peptide sequence specificity records obtained by methods of the inventions, which may be stored in the computer; (3) a comparison target, such as a query target; and (4) a program for alignment and comparison, typically with rank-ordering of comparison results on the basis of computed similarity values.

Characteristics of ovarian cancer-associated proteins

Ovarian cancer proteins of the present invention may be categorized as secreted proteins, transmembrane proteins, or intracellular proteins. In one embodiment, the ovarian cancer protein is an intracellular protein. Intracellular proteins may be found in the cytoplasm and/or in the nucleus. Intracellular proteins are involved in all aspects of cellular function and replication (including, e.g., signaling pathways); aberrant expression of such proteins often results in unregulated or dysregulated cellular processes. See, e.g., Alberts, et al. (eds. 1994) *Molecular Biology of the Cell* (3d ed.) Garland. For example, many intracellular proteins have enzymatic activity such as protein kinase activity, protein phosphatase activity, protease activity, nucleotide cyclase activity, polymerase activity, and the like. Intracellular proteins can also serve as docking proteins that are involved in organizing complexes of proteins, or targeting proteins to various subcellular localizations, and are often involved in maintaining the structural integrity of organelles.

An increasingly appreciated concept in characterizing proteins is the presence in the proteins of one or more structural motifs for which defined functions have been attributed. In addition to the highly conserved sequences found in the enzymatic domain of proteins, highly conserved sequences have been identified in proteins that are involved in protein-protein interaction. For example, Src-homology-2 (SH2) domains bind tyrosine-phosphorylated targets in a sequence dependent manner. PTB domains, which are distinct from SH2 domains, also bind tyrosine phosphorylated targets. SH3 domains bind to proline-rich targets. In addition, PH domains, tetratricopeptide repeats and WD domains to name only a few, have been shown to mediate protein-protein interactions. Some of these may also be involved in binding to phospholipids or other second messengers. As will be appreciated by one of ordinary skill in the art, these motifs can be identified on the basis of amino acid sequence; thus, an analysis of the sequence of proteins may provide insight into both the enzymatic potential of the molecule and/or molecules with which the protein may associate. One useful database is Pfam (protein families), which is a large collection of multiple sequence alignments and hidden Markov models covering many common protein domains. Versions are available via the internet from Washington University in St. Louis, the Sanger Center in England, and the Karolinska Institute in Sweden. See, e.g., Bateman, et al. (2000) *Nuc. Acids Res.* 28:263-266; Sonnhammer, et al. (1997) *Proteins* 28:405-420; Bateman, et al. (1999) *Nuc. Acids Res.* 27:260-262; and Sonnhammer, et al. (1998) *Nuc. Acids Res.* 26:320-

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In another preferred embodiment, the ovarian cancer sequences are transmembrane proteins. Transmembrane proteins are molecules that span a phospholipid bilayer of a cell. They may have an intracellular domain, an extracellular domain, or both. The intracellular domains of such proteins may have a number of functions including those already described for intracellular proteins. For example, the intracellular domain may have enzymatic activity and/or may serve as a binding site for additional proteins. Frequently the intracellular domain of transmembrane proteins serves both roles. For example certain receptor tyrosine kinases have both protein kinase activity and SH2 domains. In addition, autophosphorylation of tyrosines on the receptor molecule itself, creates binding sites for additional SH2 domain containing proteins.

Transmembrane proteins may contain from one to many transmembrane domains. For example, receptor tyrosine kinases, certain cytokine receptors, receptor guanylyl cyclases and receptor serine/threonine protein kinases contain a single transmembrane domain. However, various other proteins including channels and adenylyl cyclases contain numerous transmembrane domains. Many important cell surface receptors such as G protein coupled receptors (GPCRs) are classified as "seven transmembrane domain" proteins, as they contain 7 membrane spanning regions. Characteristics of transmembrane domains include approximately 17 consecutive hydrophobic amino acids that may be followed by charged amino acids. Therefore, upon analysis of the amino acid sequence of a particular protein, the localization and number of transmembrane domains within the protein may be predicted (see, e.g., PSORT web site <http://psort.nibb.ac.jp/>). Important transmembrane protein receptors include, but are not limited to the insulin receptor, insulin-like growth factor receptor, human growth hormone receptor, glucose transporters, transferrin receptor, epidermal growth factor receptor, low density lipoprotein receptor, epidermal growth factor receptor, leptin receptor, interleukin receptors, e.g., IL-1 receptor, IL-2 receptor, etc.

The extracellular domains of transmembrane proteins are diverse; however, conserved motifs are found repeatedly among various extracellular domains. Conserved structure and/or functions have been ascribed to different extracellular motifs. Many extracellular domains are involved in binding to other molecules. In one aspect, extracellular domains are found on receptors. Factors that bind the receptor domain include circulating ligands, which may be peptides, proteins, or small molecules such as adenosine and the like. For example, growth factors such as EGF, FGF, and PDGF are circulating growth factors that bind to their

cognate receptors to initiate a variety of cellular responses. Other factors include cytokines, mitogenic factors, neurotrophic factors and the like. Extracellular domains also bind to cell-associated molecules, or may be processed or shed to the blood stream. In this respect, they can mediate cell-cell interactions. Cell-associated ligands can be tethered to the cell, e.g., via a glycosylphosphatidylinositol (GPI) anchor, or may themselves be transmembrane proteins. Extracellular domains also associate with the extracellular matrix and contribute to the maintenance of the cell structure.

Ovarian cancer proteins that are transmembrane are particularly preferred in the present invention as they are readily accessible targets for immunotherapeutics, as are described herein. In addition, as outlined below, transmembrane proteins can be also useful in imaging modalities. Antibodies may be used to label such readily accessible proteins *in situ*. Alternatively, antibodies can also label intracellular proteins, in which case samples are typically permeabilized to provide access to intracellular proteins. In addition, some membrane proteins can be processed to release a soluble protein, or to expose a residual fragment. Released soluble proteins may be useful diagnostic markers, processed residual protein fragments may be useful ovarian markers of disease.

It will also be appreciated by those in the art that a transmembrane protein can be made soluble by removing transmembrane sequences, e.g., through recombinant methods. Furthermore, transmembrane proteins that have been made soluble can be made to be secreted through recombinant means by adding an appropriate signal sequence.

In another embodiment, the ovarian cancer proteins are secreted proteins; the secretion of which can be either constitutive or regulated. These proteins may have a signal peptide or signal sequence that targets the molecule to the secretory pathway. Secreted proteins are involved in numerous physiological events; e.g., if circulating, they often serve to transmit signals to various other cell types. The secreted protein may function in an autocrine manner (acting on the cell that secreted the factor), a paracrine manner (acting on cells in close proximity to the cell that secreted the factor), an endocrine manner (acting on cells at a distance, e.g., secretion into the blood stream), or exocrine (secretion, e.g., through a duct or to an adjacent epithelial surface as sweat glands, sebaceous glands, pancreatic ducts, lacrimal glands, mammary glands, wax producing glands of the ear, etc.). Thus, secreted molecules often find use in modulating or altering numerous aspects of physiology. Ovarian cancer proteins that are secreted proteins are particularly preferred as good diagnostic markers, e.g., for blood, plasma, serum, or stool tests. Those which are enzymes may be

antibody or small molecule therapeutic targets. Others may be useful as vaccine targets, e.g., via CTL mechanisms, as protein or DNA vaccines.

Use of ovarian cancer nucleic acids

As described above, ovarian cancer sequence is initially identified by substantial nucleic acid and/or amino acid sequence homology or linkage to the ovarian cancer sequences outlined herein. Such homology can be based upon the overall nucleic acid or amino acid sequence, and is generally determined as outlined below, using either homology programs or hybridization conditions. Typically, linked sequences on a mRNA are found on the same molecule.

The ovarian cancer nucleic acid sequences of the invention, e.g., in Table 1-26, can be fragments of larger genes, e.g., they are nucleic acid segments. "Genes" in this context includes coding regions, non-coding regions, and mixtures of coding and non-coding regions. Accordingly, as will be appreciated by those in the art, using the sequences provided herein, extended sequences, in either direction, of the ovarian cancer genes can be obtained, using techniques well known in the art for cloning either longer sequences or the full length sequences; see Ausubel, et al., *supra*. Much can be done by informatics and many sequences can be clustered to include multiple sequences corresponding to a single gene, e.g., systems such as UniGene (see, <http://www.ncbi.nlm.nih.gov/UniGene/>).

Once the ovarian cancer nucleic acid is identified, it can be cloned and, if necessary, its constituent parts recombined to form the entire ovarian cancer nucleic acid coding regions or the entire mRNA sequence. Once isolated from its natural source, e.g., contained within a plasmid or other vector or excised as a linear nucleic acid segment, the recombinant ovarian cancer nucleic acid can be further-used as a probe to identify and isolate other ovarian cancer nucleic acids, e.g., extended coding regions. It can also be used as a "precursor" nucleic acid to make modified or variant ovarian cancer nucleic acids and proteins.

The ovarian cancer nucleic acids of the present invention are useful in several ways. In a first embodiment, nucleic acid probes to the ovarian cancer nucleic acids are made and attached to biochips to be used in screening and diagnostic methods, as outlined below, or for administration, e.g., for gene therapy, vaccine, RNAi, and/or antisense applications. Alternatively, the ovarian cancer nucleic acids that include coding regions of ovarian cancer proteins can be put into expression vectors for the expression of ovarian cancer proteins, again for screening purposes or for administration to a patient.

In a preferred embodiment, nucleic acid probes to ovarian cancer nucleic acids (both the nucleic acid sequences outlined in the figures and/or the complements thereof) are made.

The nucleic acid probes attached to the biochip are designed to be substantially complementary to the ovarian cancer nucleic acids, e.g., the target sequence (either the target sequence of the sample or to other probe sequences, e.g., in sandwich assays), such that hybridization of the target sequence and the probes of the present invention occurs. As outlined below, this complementarity need not be perfect; there may be any number of base pair mismatches which will interfere with hybridization between the target sequence and the single stranded nucleic acids of the present invention. However, if the number of mutations is so great that no hybridization can occur under even the least stringent of hybridization conditions, the sequence is not a complementary target sequence. Thus, by "substantially complementary" herein is meant that the probes are sufficiently complementary to the target sequences to hybridize under normal reaction conditions, particularly high stringency conditions, as outlined herein.

A nucleic acid probe is generally single stranded but can be partially single and partially double stranded. The strandedness of the probe is dictated by the structure, composition, and properties of the target sequence. In general, the nucleic acid probes range from about 8 to about 100 bases long, with from about 10 to about 80 bases being preferred, and from about 30 to about 50 bases being particularly preferred. That is, generally whole genes are not used. In some embodiments, much longer nucleic acids can be used, up to hundreds of bases.

In a preferred embodiment, more than one probe per sequence is used, with either overlapping probes or probes to different sections of the target being used. That is, two, three, four or more probes, with three being preferred, are used to build in a redundancy for a particular target. The probes can be overlapping (e.g., have some sequence in common), or separate. In some cases, PCR primers may be used to amplify signal for higher sensitivity.

As will be appreciated by those in the art, nucleic acids can be attached or immobilized to a solid support in a wide variety of ways. By "immobilized" and grammatical equivalents herein is meant the association or binding between the nucleic acid probe and the solid support is sufficient to be stable under the conditions of binding, washing, analysis, and removal as outlined below. The binding can typically be covalent or non-covalent. By "non-covalent binding" and grammatical equivalents herein is meant one or more of electrostatic, hydrophilic, and hydrophobic interactions. Included in non-covalent binding is the covalent

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attachment of a molecule, such as, streptavidin to the support and the non-covalent binding of the biotinylated probe to the streptavidin. By "covalent binding" and grammatical equivalents herein is meant that the two moieties, the solid support and the probe, are attached by at least one bond, including sigma bonds, pi bonds and coordination bonds.

Covalent bonds can be formed directly between the probe and the solid support or can be formed by a cross linker or by inclusion of a specific reactive group on either the solid support or the probe or both molecules. Immobilization may also involve a combination of covalent and non-covalent interactions.

In general, the probes are attached to the biochip in a wide variety of ways, as will be appreciated by those in the art. As described herein, the nucleic acids can either be synthesized first, with subsequent attachment to the biochip, or can be directly synthesized on the biochip.

The biochip comprises a suitable solid substrate. By "substrate" or "solid support" or other grammatical equivalents herein is meant a material that can be modified to contain discrete individual sites appropriate for the attachment or association of the nucleic acid probes and is amenable to at least one detection method. As will be appreciated by those in the art, the number of possible substrates are very large, and include, but are not limited to, glass and modified or functionalized glass, plastics (including acrylics, polystyrene and copolymers of styrene and other materials, polypropylene, polyethylene, polybutylene, polyurethanes, Teflon, etc.), polysaccharides, nylon or nitrocellulose, resins, silica or silica-based materials including silicon and modified silicon, carbon, metals, inorganic glasses, plastics, etc. In general, the substrates allow optical detection and do not appreciably fluoresce. See, e.g., WO0055627 Reusable Low Fluorescent Plastic Biochip.

Generally the substrate is planar, although as will be appreciated by those in the art, other configurations of substrates may be used as well. For example, the probes may be placed on the inside surface of a tube, for flow-through sample analysis to minimize sample volume. Similarly, the substrate may be flexible, such as a flexible foam, including closed cell foams made of particular plastics.

In a preferred embodiment, the surface of the biochip and the probe may be derivatized with chemical functional groups for subsequent attachment of the two. Thus, e.g., the biochip is derivatized with a chemical functional group including, but not limited to, amino groups, carboxyl groups, oxo groups and thiol groups, with amino groups being particularly preferred. Using these functional groups, the probes can be attached using

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functional groups on the probes. For example, nucleic acids containing amino groups can be attached to surfaces comprising amino groups, e.g., using linkers as are known in the art; e.g., homo- or hetero-bifunctional linkers as are well known (see 1994 Pierce Chemical Company catalog, technical section on cross-linkers, pages 155-200). In addition, in some cases, additional linkers, such as alkyl groups (including substituted and heteroalkyl groups) may be used.

In this embodiment, oligonucleotides are synthesized as is known in the art, and then attached to the surface of the solid support. As will be appreciated by those skilled in the art, either the 5' or 3' terminus may be attached to the solid support, or attachment may be via an internal nucleoside.

In another embodiment, the immobilization to the solid support may be very strong, yet non-covalent. For example, biotinylated oligonucleotides can be made, which bind to surfaces covalently coated with streptavidin, resulting in attachment.

Alternatively, the oligonucleotides may be synthesized on the surface, as is known in the art. For example, photoactivation techniques utilizing photopolymerization compounds and techniques are used. In a preferred embodiment, the nucleic acids can be synthesized in situ, using well known photolithographic techniques, such as those described in WO 95/25116; WO 95/35505; U.S. Patent Nos. 5,700,637 and 5,445,934; and references cited within, all of which are expressly incorporated by reference; these methods of attachment form the basis of the Affymetrix GeneChip™ technology.

Often, amplification-based assays are performed to measure the expression level of ovarian cancer-associated sequences. These assays are typically performed in conjunction with reverse transcription. In such assays, an ovarian cancer-associated nucleic acid sequence acts as a template in an amplification reaction (e.g., Polymerase Chain Reaction, or PCR). In a quantitative amplification, the amount of amplification product will be proportional to the amount of template in the original sample. Comparison to appropriate controls provides a measure of the amount of ovarian cancer-associated RNA. Methods of quantitative amplification are well known to those of skill in the art. Detailed protocols for quantitative PCR are available. See, e.g., Innis, et al (1990) PCR Protocols: A Guide to Methods and Applications Academic Press.

In some embodiments, a TaqMan based assay is used to measure expression. TaqMan based assays use a fluorogenic oligonucleotide probe that contains a 5' fluorescent dye and a 3' quenching agent. The probe hybridizes to a PCR product, but cannot itself be

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extended due to a blocking agent at the 3' end. When the PCR product is amplified in subsequent cycles, the 5' nuclease activity of the polymerase, e.g., AmpliTaq, results in the cleavage of the TaqMan probe. This cleavage separates the 5' fluorescent dye and the 3' quenching agent, thereby resulting in an increase in fluorescence as a function of amplification (see, e.g., literature provided by Perkin-Elmer, e.g., www2.perkin-elmer.com).

Other suitable amplification methods include, but are not limited to, ligase chain reaction (LCR; see Wu and Wallace (1989) Genomics 4:560-569; Landegren, et al. (1988) Science 241:1077-1080; and Barringer, et al. (1990) Gene 89:117-122), transcription amplification (Kwoh, et al. (1989) Proc. Natl. Acad. Sci. USA 86:1173-1177), self-sustained sequence replication (Guatelli, et al. (1990) Proc. Natl. Acad. Sci. USA 87:1874-1878), dot PCR, linker adapter PCR, etc.

Expression of ovarian cancer proteins from nucleic acids

In a preferred embodiment, ovarian cancer nucleic acids, e.g., encoding ovarian cancer proteins are used to make a variety of expression vectors to express ovarian cancer proteins which can then be used in screening assays, as described below. Expression vectors and recombinant DNA technology are well known and are used to express proteins. See, e.g., Ausubel, supra; and Fernandez and Hoeffler (eds. 1999) Gene Expression Systems Academic Press. The expression vectors may be either self-replicating extrachromosomal vectors or vectors which integrate into a host genome. Generally, these expression vectors include transcriptional and translational regulatory nucleic acid operably linked to the nucleic acid encoding the ovarian cancer protein. The term "control sequences" refers to DNA sequences used for the expression of an operably linked coding sequence in a particular host organism. Control sequences that are suitable for prokaryotes, e.g., include a promoter, optionally an operator sequence, and a ribosome binding site. Eukaryotic cells are known to utilize promoters, polyadenylation signals, and enhancers.

Nucleic acid is "operably linked" when it is placed into a functional relationship with another nucleic acid sequence. For example, DNA for a pre-sequence or secretory leader is operably linked to DNA for a polypeptide if it is expressed as a pre-protein that participates in the secretion of the polypeptide; a promoter or enhancer is operably linked to a coding sequence if it affects the transcription of the sequence; a ribosome binding site is operably linked to a coding sequence if it is positioned so as to facilitate translation; and two sequences may be operably linked when they are physically part of the same polymer. Generally,

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“operably linked” means that the DNA sequences being linked are contiguous, and, in the case of a secretory leader, contiguous and in reading phase. However, enhancers do not have to be contiguous. Linking is typically accomplished by ligation at convenient restriction sites. If such sites do not exist, synthetic oligonucleotide adaptors or linkers are used in accordance with conventional practice. Transcriptional and translational regulatory nucleic acid will generally be appropriate to the host cell used to express the ovarian cancer protein. Numerous types of appropriate expression vectors, and suitable regulatory sequences are known in the art for a variety of host cells.

In general, transcriptional and translational regulatory sequences may include, but are not limited to, promoter sequences, ribosomal binding sites, transcriptional start and stop sequences, translational start and stop sequences, and enhancer or activator sequences. In a preferred embodiment, the regulatory sequences include a promoter and transcriptional start and stop sequences.

Promoter sequences typically encode constitutive or inducible promoters. The promoters may be naturally occurring promoters or hybrid promoters. Hybrid promoters, which combine elements of more than one promoter, are also known in the art, and are useful in the present invention.

In addition, an expression vector may comprise additional elements. For example, the expression vector may have two replication systems, thus allowing it to be maintained in two organisms, e.g., in mammalian or insect cells for expression and in a procaryotic host for cloning and amplification. Furthermore, for integrating expression vectors, the expression vector contains at least one sequence homologous to the host cell genome, and preferably two homologous sequences which flank the expression construct. The integrating vector may be directed to a specific locus in the host cell by selecting the appropriate homologous sequence for inclusion in the vector. Constructs for integrating vectors are available. See, e.g., Fernandez and Hoeffler, *supra*.

In addition, in a preferred embodiment, the expression vector contains a selectable marker gene to allow the selection of transformed host cells. Selection genes are well known in the art and will vary with the host cell used.

The ovarian cancer proteins of the present invention are produced by culturing a host cell transformed with an expression vector containing nucleic acid encoding an ovarian cancer protein, under the appropriate conditions to induce or cause expression of the ovarian cancer protein. Conditions appropriate for ovarian cancer protein expression will vary with

the choice of the expression vector and the host cell, and will be easily ascertained by one skilled in the art through routine experimentation or optimization. For example, the use of constitutive promoters in the expression vector will require optimizing the growth and proliferation of the host cell, while the use of an inducible promoter requires the appropriate growth conditions for induction. In addition, in some embodiments, the timing of the harvest is important. For example, the baculovirus systems used in insect cell expression are lytic viruses, and thus harvest time selection can be crucial for product yield.

Appropriate host cells include yeast, bacteria, archaeobacteria, fungi, and insect and animal cells, including mammalian cells. Of particular interest are *Saccharomyces cerevisiae* and other yeasts, *E. coli*, *Bacillus subtilis*, Sf9 cells, C129 cells, 293 cells, Neurospora, BHK, CHO, COS, HeLa cells, HUVEC (human umbilical vein endothelial cells), THP1 cells (a macrophage cell line) and various other human cells and cell lines.

In a preferred embodiment, the ovarian cancer proteins are expressed in mammalian cells. Mammalian expression systems are also known in the art, and include retroviral and adenoviral systems. One expression vector system is a retroviral vector system such as is generally described in PCT/US97/01019 and PCT/US97/01048, both of which are hereby expressly incorporated by reference. Of particular use as mammalian promoters are the promoters from mammalian viral genes, since the viral genes are often highly expressed and have a broad host range. Examples include the SV40 early promoter, mouse mammary tumor virus LTR promoter, adenovirus major late promoter, herpes simplex virus promoter, and the CMV promoter. See, e.g., Fernandez and Hoeffler, *supra*. Typically, transcription termination and polyadenylation sequences recognized by mammalian cells are regulatory regions located 3' to the translation stop codon and thus, together with the promoter elements, flank the coding sequence. Examples of transcription terminator and polyadenylation signals include those derived from SV40.

The methods of introducing exogenous nucleic acid into mammalian hosts, as well as other hosts, is well known in the art, and will vary with the host cell used. Techniques include dextran-mediated transfection, calcium phosphate precipitation, polybrene mediated transfection, protoplast fusion, electroporation, viral infection, encapsulation of the polynucleotide(s) in liposomes, and direct microinjection of the DNA into nuclei.

In a preferred embodiment, ovarian cancer proteins are expressed in bacterial systems. Bacterial expression systems are well known in the art. Promoters from bacteriophage may also be used and are known in the art. In addition, synthetic promoters and hybrid promoters

are also useful; e.g., the tac promoter is a hybrid of the trp and lac promoter sequences.

Furthermore, a bacterial promoter can include naturally occurring promoters of non-bacterial origin that have the ability to bind bacterial RNA polymerase and initiate transcription. In addition to a functioning promoter sequence, an efficient ribosome binding site is desirable.

5 The expression vector may also include a signal peptide sequence that provides for secretion of the ovarian cancer protein in bacteria. The protein is either secreted into the growth media (gram-positive bacteria) or into the periplasmic space, located between the inner and outer membrane of the cell (gram-negative bacteria). The bacterial expression vector may also include a selectable marker gene to allow for the selection of bacterial strains that have been transformed. Suitable selection genes include genes which render the bacteria resistant to drugs such as ampicillin, chloramphenicol, erythromycin, kanamycin, neomycin, and tetracycline. Selectable markers also include biosynthetic genes, such as those in the histidine, tryptophan, and leucine biosynthetic pathways. These components are assembled into expression vectors. Expression vectors for bacteria are well known in the art, and include vectors for *Bacillus subtilis*, *E. coli*, *Streptococcus cremoris*, and *Streptococcus lividans*, among others. See Fernandez and Hoeffler, supra. The bacterial expression vectors are transformed into bacterial host cells using techniques well known in the art, such as calcium chloride treatment, electroporation, and others.

15 In one embodiment, ovarian cancer proteins are produced in insect cells. Expression vectors for the transformation of insect cells, and in particular, baculovirus-based expression vectors, are well known in the art.

In a preferred embodiment, an ovarian cancer protein is produced in yeast cells.

25 Yeast expression systems are well known in the art, and include expression vectors for *Saccharomyces cerevisiae*, *Candida albicans* and *C. maltosa*, *Hansenula polymorpha*, *Kluyveromyces fragilis* and *K. lactis*, *Pichia guilliermondii* and *P. pastoris*, *Schizosaccharomyces pombe*, and *Yarrowia lipolytica*.

30 The ovarian cancer protein may also be made as a fusion protein, using techniques well known in the art. Thus, e.g., for the creation of monoclonal antibodies, if the desired epitope is small, the ovarian cancer protein may be fused to a carrier protein to form an immunogen. Alternatively, the ovarian cancer protein may be made as a fusion protein to increase expression, or for other reasons. For example, when the ovarian cancer protein is an ovarian cancer peptide, the nucleic acid encoding the peptide may be linked to other nucleic acid for expression purposes.

In a preferred embodiment, the ovarian cancer protein is purified or isolated after expression. Ovarian cancer proteins may be isolated or purified in a variety of ways known to those skilled in the art depending on what other components are present in the sample.

5 Standard purification methods include electrophoretic, molecular, immunological and chromatographic techniques, including ion exchange, hydrophobic, affinity, and reverse-phase HPLC chromatography, and chromatofocusing. For example, the ovarian cancer protein may be purified using a standard anti-ovarian cancer protein antibody column.

Ultrafiltration and diafiltration techniques, in conjunction with protein concentration, are also useful. For general guidance in suitable purification techniques, see Scopes (1982) *Protein*

10 *Purification* Springer-Verlag. The degree of purification necessary will vary depending on the use of the ovarian cancer protein. In some instances no purification will be necessary.

Once expressed and purified if necessary, the ovarian cancer proteins and nucleic acids are useful in a number of applications. They may be used as immunoselection reagents, as vaccine reagents, as screening agents, etc.

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Variants of ovarian cancer proteins

In one embodiment, the ovarian cancer proteins are derivative or variant ovarian cancer proteins as compared to the wild-type sequence. That is, as outlined more fully below, the derivative ovarian cancer peptide will often contain at least one amino acid substitution, deletion or insertion, with amino acid substitutions being particularly preferred. The amino acid substitution, insertion, or deletion may occur at most any residue within the ovarian cancer peptide.

Also included within one embodiment of ovarian cancer proteins of the present invention are amino acid sequence variants. These variants typically fall into one or more of three classes: substitutional, insertional or deletional variants. These variants ordinarily are prepared by site specific mutagenesis of nucleotides in the DNA encoding the ovarian cancer protein, using cassette or PCR mutagenesis or other techniques well known in the art, to produce DNA encoding the variant, and thereafter expressing the DNA in recombinant cell culture as outlined above. However, variant ovarian cancer protein fragments having up to about 100-150 residues may be prepared by in vitro synthesis using established techniques.

30 Amino acid sequence variants are characterized by the predetermined nature of the variation, a feature that sets them apart from naturally occurring allelic or interspecies variation of the ovarian cancer protein amino acid sequence. The variants typically exhibit the same

qualitative biological activity as the naturally occurring analogue, although variants can also be selected which have modified characteristics as will be more fully outlined below.

While the site or region for introducing an amino acid sequence variation is

predetermined, the mutation per se need not be predetermined. For example, in order to optimize the performance of a mutation at a given site, random mutagenesis may be conducted at the target codon or region and the expressed ovarian cancer variants screened for the optimal combination of desired activity. Techniques for making substitution mutations at predetermined sites in DNA having a known sequence are well known, e.g., M13 primer mutagenesis and PCR mutagenesis. Screening of the mutants is done using assays of ovarian cancer protein activities.

Amino acid substitutions are typically of single residues; insertions usually will be on the order of from about 1 to 20 amino acids, although considerably larger insertions may be tolerated. Deletions range from about 1 to about 20 residues, although in some cases deletions may be much larger.

Substitutions, deletions, insertions or any combination thereof may be used to arrive at a final derivative. Generally these changes are done on a few amino acids to minimize the alteration of the molecule. However, larger changes may be tolerated in certain circumstances. When small alterations in the characteristics of the ovarian cancer protein are desired, substitutions are generally made in accordance with the amino acid substitution relationships provided in the definition section.

The variants typically exhibit the same qualitative biological activity and will elicit the same immune response as the naturally-occurring analog, although variants also are selected to modify the characteristics of the ovarian cancer proteins as needed. Alternatively, the variant may be designed such that the biological activity of the ovarian cancer protein is altered. For example, glycosylation sites may be altered or removed.

Substantial changes in function or immunological identity are made by selecting substitutions that are less conservative than those described above. For example,

substitutions may be made which more significantly affect the structure of the polypeptide backbone in the area of the alteration, for example the alpha-helical or beta-sheet structure; the charge or hydrophobicity of the molecule at the target site; or the bulk of the side chain.

The substitutions which in general are expected to produce the greatest changes in the polypeptide's properties are those in which (a) a hydrophilic residue, e.g., serine or threonine is substituted for (or by) a hydrophobic residue, e.g., leucine, isoleucine, phenylalanine,

valine, or alanine; (b) a cysteine or proline is substituted for (or by) any other residue; (c) a residue having an electropositive side chain, e.g., lysine, arginine, or histidine, is substituted for (or by) an electronegative residue, e.g., glutamic or aspartic acid; (d) a residue having a bulky side chain, e.g., phenylalanine, is substituted for (or by) one not having a side chain, e.g., glycine; or (e) a proline residue is incorporated or substituted, which changes the degree of rotational freedom of the peptidyl bond.

Covalent modifications of ovarian cancer polypeptides are included within the scope of this invention. One type of covalent modification includes reacting targeted amino acid residues of an ovarian cancer polypeptide with an organic derivatizing agent that is capable of reacting with selected side chains or the N-or C-terminal residues of an ovarian cancer polypeptide. Derivatization with bifunctional agents is useful, for instance, for crosslinking ovarian cancer polypeptides to a water-insoluble support matrix or surface for use in the method for purifying anti-ovarian cancer polypeptide antibodies or screening assays, as is more fully described below. Commonly used crosslinking agents include, e.g., 1,1-

bis(diazoacetyl)-2-phenylethane, glutaraldehyde, N-hydroxysuccinimide esters, e.g., esters with 4-azidosalicylic acid, homobifunctional imidoesters, including disuccinimidyl esters such as 3,3'-dithiobis(succinimidylpropionate), bifunctional maleimides such as bis-N-maleimido-1,8-octane and agents such as methyl-3-(p-azidophenyl)dithio)propionimide.

Other modifications include deamidation of glutamine and asparagine residues to the corresponding glutamic and aspartic acid residues, respectively, hydroxylation of proline and lysine, phosphorylation of hydroxyl groups of serine, threonine, or tyrosine residues, methylation of the amino groups of the lysine, arginine, and histidine side chains (e.g., pp. 79-86, Creighton (1983) Proteins: Structure and Molecular Properties Freeman), acetylation of the N-terminal amine, and amidation of a C-terminal carboxyl group.

Another type of covalent modification of the ovarian cancer polypeptide included within the scope of this invention comprises altering the native glycosylation pattern of the polypeptide. "Altering the native glycosylation pattern" is intended for purposes herein to mean deleting one or more carbohydrate moieties found in native sequence ovarian cancer polypeptide, and/or adding one or more glycosylation sites that are not present in the native sequence ovarian cancer polypeptide. Glycosylation patterns can be altered in many ways. For example the use of different cell types to express ovarian cancer-associated sequences can result in different glycosylation patterns.

Addition of glycosylation sites to ovarian cancer polypeptides may also be

accomplished by altering the amino acid sequence thereof. The alteration may be made, e.g., by the addition of, or substitution by, one or more serine or threonine residues to the native sequence ovarian cancer polypeptide (for O-linked glycosylation sites). The ovarian cancer amino acid sequence may optionally be altered through changes at the DNA level,

5 particularly by mutating the DNA encoding the ovarian cancer polypeptide at pre-selected bases such that codons are generated that will translate into the desired amino acids.

Another means of increasing the number of carbohydrate moieties on the ovarian cancer polypeptide is by chemical or enzymatic coupling of glycosides to the polypeptide. See, e.g., WO 87/05330, and pp. 259-306 in Aplin and Wriston (1981) CRC Crit. Rev. Biochem. CRC Press.

10 Removal of carbohydrate moieties present on the ovarian cancer polypeptide may be accomplished chemically or enzymatically or by mutational substitution of codons encoding for amino acid residues that serve as targets for glycosylation. Chemical deglycosylation techniques are applicable. See, e.g., Sojar and Bahl (1987) Arch. Biochem. Biophys. 259:52-57; and Edge, et al. (1981) Anal. Biochem. 118:131-137. Enzymatic cleavage of carbohydrate moieties on polypeptides can be achieved by the use of a variety of endo- and exo-glycosidases. See, e.g., Thotakura, et al. (1987) Meth. Enzymol. 138:350-359.

Another type of covalent modification of ovarian cancer comprises linking the ovarian cancer polypeptide to one of a variety of non-proteinaceous polymers, e.g., polyethylene glycol, polypropylene glycol, or polyoxyalkylene. See, e.g., U.S. Patent Nos. 4,640,835; 4,496,689; 4,301,144; 4,670,417; 4,791,192; or 4,179,337.

Ovarian cancer polypeptides of the present invention may also be modified in a way to form chimeric molecules, e.g., comprising an ovarian cancer polypeptide fused to another heterologous polypeptide or amino acid sequence. In one embodiment, such a chimeric molecule comprises a fusion of an ovarian cancer polypeptide with a tag polypeptide which provides an epitope to which an anti-tag antibody can selectively bind. The epitope tag is generally placed at the amino- or carboxyl-terminus of the ovarian cancer polypeptide. The presence of such epitope-tagged forms of an ovarian cancer polypeptide can be detected using an antibody against the tag polypeptide. Also, provision of the epitope tag enables the ovarian cancer polypeptide to be readily purified by affinity purification using an anti-tag antibody or another type of affinity matrix that binds to the epitope tag. In an alternative embodiment, the chimeric molecule may comprise a fusion of an ovarian cancer polypeptide with an immunoglobulin or a particular region of an immunoglobulin. For a bivalent form of

the chimeric molecule, such a fusion could be to the Fc region of an IgG molecule.

Various tag polypeptides and their respective antibodies are well known in the art. Examples include poly-histidine (poly-his) or poly-histidine-glycine (poly-his-gly) tags; His6 and metal chelation tags, the flu HA tag polypeptide and its antibody 12CA5 (Field, et al. (1988) Mol. Cell. Biol. 8:2159-2165), the c-myc tag and the 8F9, 3C7, 6E10, G4, B7, and 9E10 antibodies thereto (Evan, et al. (1985) Mol. Cell. Biol. 5:3610-3616), and the Herpes Simplex virus glycoprotein D (gD) tag and its antibody (Paborsky, et al. (1990) Protein Engineering 3:547-553). Other tag polypeptides include, e.g., the Flag-peptide (Hopp, et al. (1988) BioTechnology 6:1204-1210), the KT3 epitope peptide (Martin, et al. (1992) Science 255:192-194), tubulin epitope peptide (Skinner, et al. (1991) J. Biol. Chem. 266:15163-15166), and the T7 gene 10 protein peptide tag (Lutz-Freyermuth et al. (1990) Proc. Natl. Acad. Sci. USA 87:6393-6397).

Also included are other ovarian cancer proteins of the ovarian cancer family, and ovarian cancer proteins from other organisms, which are cloned and expressed as outlined below. Thus, probe or degenerate polymerase chain reaction (PCR) primer sequences may be used to find other related ovarian cancer proteins from humans or other organisms. As will be appreciated by those in the art, particularly useful probe and/or PCR primer sequences include the unique areas of the ovarian cancer nucleic acid sequence. As is generally known in the art, preferred PCR primers are from about 15 to about 35 nucleotides in length, with from about 20 to about 30 being preferred, and may contain inosine as needed. The conditions for the PCR reaction are well known in the art (e.g., Innis, PCR Protocols, supra).

Antibodies to ovarian cancer proteins

In a preferred embodiment, when the ovarian cancer protein is to be used to generate antibodies, e.g., for immunotherapy or immunodiagnosis, the ovarian cancer protein should share at least one epitope or determinant with the full length protein. By "epitope" or "determinant" herein is typically meant a portion of a protein which will generate and/or bind an antibody or T-cell receptor in the context of MHC. Thus, in most instances, antibodies made to a smaller ovarian cancer protein will be able to bind to the full-length protein, particularly linear epitopes. In a preferred embodiment, the epitope is unique; that is, antibodies generated to a unique epitope show little or no cross-reactivity.

Methods of preparing polyclonal antibodies are known to the skilled artisan (e.g., Coligan, supra; and Harlow and Lane, supra). Polyclonal antibodies can be raised in a

mammal, e.g., by one or more injections of an immunizing agent and, if desired, an adjuvant. Typically, the immunizing agent and/or adjuvant will be injected in the mammal by multiple subcutaneous or intraperitoneal injections. The immunizing agent may include a protein encoded by a nucleic acid of the figures or fragment thereof or a fusion protein thereof. It may be useful to conjugate the immunizing agent to a protein known to be immunogenic in the mammal being immunized. Examples of such immunogenic proteins include but are not limited to keyhole limpet hemocyanin, serum albumin, bovine thyroglobulin, and soybean trypsin inhibitor. Examples of adjuvants which may be employed include Freund's complete adjuvant and MPL-TDM adjuvant (monophosphoryl Lipid A, synthetic trehalose dicorynomycolate). The immunization protocol may be selected by one skilled in the art without undue experimentation.

The antibodies may, alternatively, be monoclonal antibodies. Monoclonal antibodies may be prepared using hybridoma methods, such as those described by Kohler and Milstein (1975) *Nature* 256:495-497. In a hybridoma method, a mouse, hamster, or other appropriate host animal, is typically immunized with an immunizing agent to elicit lymphocytes that produce or are capable of producing antibodies that will specifically bind to the immunizing agent. Alternatively, the lymphocytes may be immunized *in vitro*. The immunizing agent will typically include a polypeptide encoded by a nucleic acid of Tables 1-26 or fragment thereof, or a fusion protein thereof. Generally, either peripheral blood lymphocytes ("PBLs") are used if cells of human origin are desired, or spleen cells or lymph node cells are used if non-human mammalian sources are desired. The lymphocytes are then fused with an immortalized cell line using a suitable fusing agent, such as polyethylene glycol, to form a hybridoma cell (e.g., pp. 59-103 in Goding (1986) Monoclonal Antibodies: Principles and Practice Academic Press). Immortalized cell lines are usually transformed mammalian cells, particularly myeloma cells of rodent, bovine and human origin. Usually, rat or mouse myeloma cell lines are employed. The hybridoma cells may be cultured in a suitable culture medium that preferably contains one or more substances that inhibit the growth or survival of the unfused, immortalized cells. For example, if the parental cells lack the enzyme hypoxanthine guanine phosphoribosyl transferase (HGPRT or HPRT), the culture medium for the hybridomas typically will include hypoxanthine, aminopterin, and thymidine ("HAT medium"), which substances prevent the growth of HGPRT-deficient cells.

In one embodiment, the antibodies are bispecific antibodies. Bispecific antibodies are monoclonal, preferably human or humanized, antibodies that have binding specificities for at

least two different antigens or that have binding specificities for two epitopes on the same antigen. In one embodiment, one of the binding specificities is for a protein encoded by a nucleic acid Table 1-26 or a fragment thereof, the other one is for any other antigen, and preferably for a cell-surface protein or receptor or receptor subunit, preferably one that is tumor specific. Alternatively, tetramer-type technology may create multivalent reagents.

In a preferred embodiment, the antibodies to ovarian cancer protein are capable of reducing or eliminating a biological function of an ovarian cancer protein, as is described below. That is, the addition of anti-ovarian cancer protein antibodies (either polyclonal or preferably monoclonal) to ovarian cancer tissue (or cells containing ovarian cancer) may reduce or eliminate the ovarian cancer. Generally, at least a 25% decrease in activity, growth, size or the like is preferred, with at least about 50% being particularly preferred and about a 95-100% decrease being especially preferred.

In a preferred embodiment the antibodies to the ovarian cancer proteins are humanized antibodies (e.g., Xenerex Biosciences; Medarex, Inc.; Abgenix, Inc.; Protein Design Labs, Inc.) Humanized forms of non-human (e.g., murine) antibodies are chimeric molecules of immunoglobulins, immunoglobulin chains or fragments thereof (such as Fv, Fab, Fab', F(ab')₂ or other antigen-binding subsequences of antibodies) which contain minimal sequence derived from non-human immunoglobulin. Humanized antibodies include human immunoglobulins (recipient antibody) in which residues from a complementary determining region (CDR) of the recipient are replaced by residues from a CDR of a non-human species (donor antibody) such as mouse, rat or rabbit having the desired specificity, affinity and capacity. In some instances, Fv framework residues of the human immunoglobulin are replaced by corresponding non-human residues. Humanized antibodies may also comprise residues which are found neither in the recipient antibody nor in the imported CDR or framework sequences. In general, a humanized antibody will comprise substantially all of at least one, and typically two, variable domains, in which all or substantially all of the CDR regions correspond to those of a non-human immunoglobulin and all or substantially all of the framework (FR) regions are those of a human immunoglobulin consensus sequence. The humanized antibody optimally also will comprise at least a portion of an immunoglobulin constant region (Fc), typically that of a human immunoglobulin. Humanization can be essentially performed following the method of Winter and co-workers, e.g., by substituting rodent CDRs or CDR sequences for the

corresponding sequences of a human antibody. See, e.g., Jones, et al. (1986) Nature 321:522-525; Riechmann, et al. (1988) Nature 332:323-329; Presta (1992) Curr. Op. Struct. Biol. 2:593-596; and Verhoeyen, et al. (1988) Science 239:1534-1536). Accordingly, such humanized antibodies are chimeric antibodies (U.S. Patent No. 4,816,567), wherein substantially less than an intact human variable domain has been substituted by the corresponding sequence from a non-human species.

Human antibodies can also be produced using various techniques known in the art, including phage display libraries (see, e.g., Hoogenboom and Winter (1991) J. Mol. Biol. 227:381-388; and Marks, et al. (1991) J. Mol. Biol. 222:581-597) or human monoclonal antibodies (see, e.g., p. 77, Cöle, et al. in Reisfeld and Sell (1985) Monoclonal Antibodies and Cancer Therapy Liss; and Boerner, et al. (1991) J. Immunol. 147:86-95). Similarly, human antibodies can be made by introducing of human immunoglobulin loci into transgenic animals, e.g., mice in which the endogenous immunoglobulin genes have been partially or completely inactivated. Upon challenge, human antibody production is observed, which closely resembles that seen in humans in all respects, including gene rearrangement, assembly, and antibody repertoire. See, e.g., U.S. Patent Nos. 5,545,807; 5,545,806; 5,569,825; 5,625,126; 5,633,425; 5,661,016; Marks, et al. (1992) Bio/Technology 10:779-783; Lonberg, et al. (1994) Nature 368:856-859; Morrison (1994) Nature 368:812-13; Neuberger (1996) Nature Biotechnology 14:826 commenting on Fishwild, et al. (1996) Nature Biotechnology 14:845-51; and Lonberg and Huszar (1995) Intern. Rev. Immunol. 13:65-93.

By immunotherapy is meant treatment of ovarian cancer, e.g., with an antibody raised against ovarian cancer proteins. As used herein, immunotherapy can be passive or active. Passive immunotherapy as defined herein is the passive transfer of antibody to a recipient (patient). Active immunization is the induction of antibody and/or T-cell responses in a recipient (patient). Induction of an immune response is the result of providing the recipient with an antigen to which antibodies are raised. The antigen may be provided by injecting a polypeptide against which antibodies are desired to be raised into a recipient, or contacting the recipient with a nucleic acid capable of expressing the antigen and under conditions for expression of the antigen, leading to an immune response.

In a preferred embodiment the ovarian cancer proteins against which antibodies are raised are secreted proteins as described above. Without being bound by theory, antibodies used for treatment, bind and prevent the secreted protein from binding to its receptor, thereby

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inactivating the secreted ovarian cancer protein.

In another preferred embodiment, the ovarian cancer protein to which antibodies are raised is a transmembrane protein. Without being bound by theory, antibodies used for treatment, bind the extracellular domain of the ovarian cancer protein and prevent it from binding to other proteins, such as circulating ligands or cell-associated molecules. The antibody may cause down-regulation of the transmembrane ovarian cancer protein. As will be appreciated by one of ordinary skill in the art, the antibody may be a competitive, non-competitive or uncompetitive inhibitor of protein binding to the extracellular domain of the ovarian cancer protein. The antibody is also an antagonist of the ovarian cancer protein.

Further, the antibody prevents activation of the transmembrane ovarian cancer protein. In one aspect, when the antibody prevents the binding of other molecules to the ovarian cancer protein, the antibody prevents growth of the cell. The antibody may also be used to target or sensitize the cell to cytotoxic agents, including, but not limited to TNF- α , TNF- β , IL-1, INF- γ , and IL-2, or chemotherapeutic agents including 5FU, vinblastine, actinomycin D, cisplatin, methotrexate, and the like. In some instances the antibody belongs to a sub-type that activates serum complement when complexed with the transmembrane protein thereby mediating cytotoxicity or antigen-dependent cytotoxicity (ADCC). Thus, ovarian cancer is treated by administering to a patient antibodies directed against the transmembrane ovarian cancer protein. Antibody-labeling may activate a co-toxin, localize a toxin payload, or otherwise provide means to locally ablate cells.

In another preferred embodiment, the antibody is conjugated to an effector moiety. The effector moiety can be any number of molecules, including labeling moieties such as radioactive labels or fluorescent labels, or can be a therapeutic moiety. In one aspect the therapeutic moiety is a small molecule that modulates the activity of the ovarian cancer protein. In another aspect the therapeutic moiety modulates the activity of molecules associated with or in close proximity to the ovarian cancer protein. The therapeutic moiety may inhibit enzymatic activity such as protease or collagenase or protein kinase activity associated with ovarian cancer.

In a preferred embodiment, the therapeutic moiety can also be a cytotoxic agent. In this method, targeting the cytotoxic agent to ovarian cancer tissue or cells, results in a reduction in the number of afflicted cells, thereby reducing symptoms associated with ovarian cancer. Cytotoxic agents are numerous and varied and include, but are not limited to,

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cytotoxic drugs or toxins or active fragments of such toxins. Suitable toxins and their corresponding fragments include diphtheria A chain, exotoxin A chain, ricin A chain, abrin A chain, curcin, crotin, phenomycin, enomycin and the like. Cytotoxic agents also include radiochemicals made by conjugating radioisotopes to antibodies raised against ovarian cancer proteins, or binding of a radionuclide to a chelating agent that has been covalently attached to the antibody. Targeting the therapeutic moiety to transmembrane ovarian cancer proteins not only serves to increase the local concentration of therapeutic moiety in the ovarian cancer afflicted area, but also serves to reduce deleterious side effects that may be associated with the untargeted therapeutic moiety.

In another preferred embodiment, the ovarian cancer protein against which the antibodies are raised is an intracellular protein. In this case, the antibody may be conjugated to a protein which facilitates entry into the cell. In one case, the antibody enters the cell by endocytosis. In another embodiment, a nucleic acid encoding the antibody is administered to the individual or cell. Moreover, wherein the ovarian cancer protein can be targeted within a cell, e.g., the nucleus, an antibody thereto contains a signal for that target localization, e.g., a nuclear localization signal.

The ovarian cancer antibodies of the invention specifically bind to ovarian cancer proteins. By "specifically bind" herein is meant that the antibodies bind to the protein with a K_D of at least about 0.1 mM, more usually at least about 1 μ M, preferably at least about 0.1 μ M or better, and most preferably, 0.01 μ M or better. Selectivity of binding is also important.

Detection of ovarian cancer sequence for diagnostic and therapeutic applications

In one aspect, the RNA expression levels of genes are determined for different cellular states in the ovarian cancer phenotype. Expression levels of genes in normal tissue (e.g., not undergoing ovarian cancer) and in ovarian cancer tissue (and in some cases, for varying severities of ovarian cancer that relate to prognosis, as outlined below, or in non-malignant disease are evaluated to provide expression profiles. An expression profile of a particular cell state or point of development is essentially a "fingerprint" of the state of the cell. While two states may have any particular gene similarly expressed, the evaluation of a number of genes simultaneously allows the generation of a gene expression profile that is reflective of the state of the cell. By comparing expression profiles of cells in different states,

information regarding which genes are important (including both up- and down-regulation of genes) in each of these states is obtained. Then, diagnosis may be performed or confirmed to determine whether a tissue sample has the gene expression profile of normal or cancerous tissue. This will provide for molecular diagnosis of related conditions.

"Differential expression," or grammatical equivalents as used herein, refers to qualitative or quantitative differences in the temporal and/or cellular gene expression patterns within and among cells and tissue. Thus, a differentially expressed gene can qualitatively have its expression altered, including an activation or inactivation, in, e.g., normal versus ovarian cancer tissue. Genes may be turned on or turned off in a particular state, relative to another state thus permitting comparison of two or more states. A qualitatively regulated gene will exhibit an expression pattern within a state or cell type which is detectable by standard techniques. Some genes will be expressed in one state or cell type, but not in both. Alternatively, the difference in expression may be quantitative, e.g., in that expression is modulated, either up-regulated, resulting in an increased amount of transcript, or down-regulated, resulting in a decreased amount of transcript. The degree to which expression differs need only be large enough to quantify via standard characterization techniques as outlined below, such as by use of Affymetrix GeneChip™ expression arrays. See, e.g., Lockhart (1996) Nature Biotechnology 14:1675-1680. Other techniques include, but are not limited to, quantitative reverse transcriptase PCR, northern analysis, and RNase protection. As outlined above, preferably the change in expression (e.g., up-regulation or down-regulation) is at least about 50%, more preferably at least about 100%, more preferably at least about 150%, more preferably at least about 200%, with from 300 to at least 1000% being especially preferred.

Evaluation may be at the gene transcript, or the protein level. The amount of gene expression may be monitored using nucleic acid probes to the DNA or RNA equivalent of the gene transcript, and the quantification of gene expression levels, or, alternatively, the final gene product itself (protein) can be monitored, e.g., with antibodies to the ovarian cancer protein and standard immunoassays (ELISAs, etc.) or other techniques, including mass spectroscopy assays, 2D gel electrophoresis assays, etc. Proteins corresponding to ovarian cancer genes, e.g., those identified as being important in an ovarian cancer or disease phenotype, can be evaluated in an ovarian disease diagnostic test. In a preferred embodiment, gene expression monitoring is performed simultaneously on a number of genes. Multiple protein expression monitoring can be performed, or on an individual basis.

In this embodiment, the ovarian cancer nucleic acid probes are attached to biochips as outlined herein for the detection and quantification of ovarian cancer sequences in a particular sample. The assays are further described below in the example. PCR techniques can be used to provide greater sensitivity.

In a preferred embodiment nucleic acids encoding the ovarian cancer protein are detected. Although DNA or RNA encoding the ovarian cancer protein may be detected, of particular interest are methods wherein an mRNA encoding an ovarian cancer protein is detected. Probes to detect mRNA can be a nucleotide/deoxynucleotide probe that is complementary to and hybridizes with the mRNA and includes, but is not limited to, oligonucleotides, cDNA or RNA. Probes also should contain a detectable label, as defined herein. In one method the mRNA is detected after immobilizing the nucleic acid to be examined on a solid support such as nylon membranes and hybridizing the probe with the sample. Following washing to remove the non-specifically bound probe, the label is detected. In another method detection of the mRNA is performed *in situ*. In this method permeabilized cells or tissue samples are contacted with a detectably labeled nucleic acid probe for sufficient time to allow the probe to hybridize with the target mRNA. Following washing to remove the non-specifically bound probe, the label is detected. For example a digoxigenin labeled riboprobe (RNA probe) that is complementary to the mRNA encoding an ovarian cancer protein is detected by binding the digoxigenin with an anti-digoxigenin secondary antibody and developed with nitro blue tetrazolium and 5-bromo-4-chloro-3-indoyl phosphate.

In a preferred embodiment, various proteins from the three classes of proteins as described herein (secreted, transmembrane or intracellular proteins) are used in diagnostic assays. The ovarian cancer proteins, antibodies, nucleic acids, modified proteins and cells containing ovarian cancer sequences are used in diagnostic assays. This can be performed on an individual gene or corresponding polypeptide level. In a preferred embodiment, the expression profiles are used, preferably in conjunction with high throughput screening techniques to allow monitoring for expression profile genes and/or corresponding polypeptides.

As described and defined herein, ovarian cancer proteins, including intracellular, transmembrane, or secreted proteins, find use as prognostic or diagnostic markers of ovarian disease. Detection of these proteins in putative ovarian cancer tissue allows for detection, diagnosis, or prognosis of ovarian disease, and for selection of therapeutic strategy. In one

embodiment, antibodies are used to detect ovarian cancer proteins. A preferred method separates proteins from a sample by electrophoresis on a gel (typically a denaturing and reducing protein gel, but may be another type of gel, including isoelectric focusing gels and the like). Following separation of proteins, the ovarian cancer protein is detected, e.g., by immunoblotting with antibodies raised against the ovarian cancer protein. Methods of immunoblotting are well known to those of ordinary skill in the art.

In another preferred method, antibodies to the ovarian cancer protein find use in *in situ* imaging techniques, e.g., in histology. See, e.g., Asai (ed. 1993) Methods in Cell Biology: Antibodies in Cell Biology (vol. 37) Academic Press. Cells are contacted with from one to many antibodies to the ovarian cancer protein(s). Following washing to remove non-specific antibody binding, the presence of the antibody or antibodies is detected. In one embodiment the antibody is detected by incubating with a secondary antibody that contains a detectable label. In another method the primary antibody to the ovarian cancer protein(s) contains a detectable label, e.g., an enzyme marker that can act on a substrate. In another preferred embodiment each one of multiple primary antibodies contains a distinct and detectable label. This method finds particular use in simultaneous screening for a plurality of ovarian cancer proteins. As will be appreciated by one of ordinary skill in the art, many other histological imaging techniques are also provided by the invention.

In a preferred embodiment the label is detected in a fluorometer which has the ability to detect and distinguish emissions of different wavelengths. In addition, a fluorescence activated cell sorter (FACS) can be used in the method.

In another preferred embodiment, antibodies find use in diagnosing ovarian cancer from blood, serum, plasma, stool, and other samples. Such samples, therefore, are useful as samples to be probed or tested for the presence of ovarian cancer proteins. Antibodies can be used to detect an ovarian cancer protein by previously described immunoassay techniques including ELISA, immunoblotting (western blotting), immunoprecipitation, BIACORE technology, and the like. Conversely, the presence of antibodies may indicate an immune response against an endogenous ovarian cancer protein.

In a preferred embodiment, *in situ* hybridization of labeled ovarian cancer nucleic acid probes to tissue arrays is done. For example, arrays of tissue samples, including ovarian cancer tissue and/or normal tissue, are made. *In situ* hybridization (see, e.g., Ausubel, *supra*) is then performed. When comparing the fingerprints between an individual and a standard, the skilled artisan can make a diagnosis, a prognosis, or a prediction based on the findings. It

is further understood that the genes which indicate the diagnosis may differ from those which indicate the prognosis and molecular profiling of the condition of the cells may lead to distinctions between responsive or refractory conditions or may be predictive of outcomes.

In a preferred embodiment, the ovarian cancer proteins, antibodies, nucleic acids, modified proteins and cells containing ovarian cancer sequences are used in prognosis assays. As above, gene expression profiles can be generated that correlate to ovarian cancer, clinical, pathological, or other information, in terms of long term prognosis. Again, this may be done on either a protein or gene level, with the use of a plurality of genes being preferred. As above, ovarian cancer probes may be attached to biochips for the detection and quantification of ovarian cancer sequences in a tissue or patient. The assays proceed as outlined above for diagnosis. PCR method may provide more sensitive and accurate quantification.

Assays for therapeutic compounds

In a preferred embodiment members of the proteins, nucleic acids, and antibodies as described herein are used in drug screening assays. The ovarian cancer proteins, antibodies, nucleic acids, modified proteins and cells containing ovarian cancer sequences are used in drug screening assays or by evaluating the effect of drug candidates on a "gene expression profile" or expression profile of polypeptides. In a preferred embodiment, the expression profiles are used, preferably in conjunction with high throughput screening techniques to allow monitoring for expression profile genes after treatment with a candidate agent. See, e.g., Zlokarnik, et al. (1998) *Science* 279:84-88; and Heid (1996) *Genome Res.* 6:986-994.

In a preferred embodiment, the ovarian cancer proteins, antibodies, nucleic acids, modified proteins and cells containing the native or modified ovarian cancer proteins are used in screening assays. That is, the present invention provides novel methods for screening for compositions which modulate the ovarian cancer phenotype or an identified physiological function of an ovarian cancer protein. As above, this can be done on an individual gene level or by evaluating the effect of drug candidates on a "gene expression profile". In a preferred embodiment, the expression profiles are used, preferably in conjunction with high throughput screening techniques to allow monitoring for expression profile genes after treatment with a candidate agent. See, e.g., Zlokarnik, supra.

Having identified the differentially expressed genes herein, a variety of assays may be executed. In a preferred embodiment, assays may be run on an individual gene or protein level. That is, having identified a particular gene as up regulated in ovarian cancer, test

compounds can be screened for the ability to modulate gene expression or for binding to the ovarian cancer protein. "Modulation" thus includes both an increase and a decrease in gene expression. The preferred amount of modulation will depend on the original change of the gene expression in normal versus tissue undergoing ovarian cancer, with changes of at least 10%, preferably 50%, more preferably 100-300%, and in some embodiments 300-1000% or greater. Thus, if a gene exhibits a 4-fold increase in ovarian cancer tissue compared to normal tissue, a decrease of about four-fold is often desired; similarly, a 10-fold decrease in ovarian cancer tissue compared to normal tissue often provides a target value of a 10-fold increase in expression to be induced by the test compound.

The amount of gene expression may be monitored using nucleic acid probes and the quantification of gene expression levels, or, alternatively, the gene product itself can be monitored, e.g., through the use of antibodies to the ovarian cancer protein and standard immunoassays. Proteomics and separation techniques may also allow quantification of expression.

In a preferred embodiment, gene expression or protein monitoring of a number of entities, e.g., an expression profile, is monitored simultaneously. Such profiles will typically involve a plurality of those entities described herein.

In this embodiment, the ovarian cancer nucleic acid probes are attached to biochips as outlined herein for the detection and quantification of ovarian cancer sequences in a particular cell. Alternatively, PCR may be used. Thus, a series, e.g., of microtiter plate, may be used with dispensed primers in desired wells. A PCR reaction can then be performed and analyzed for each well.

Expression monitoring can be performed to identify compounds that modify the expression of one or more ovarian cancer-associated sequences, e.g., a polynucleotide sequence set out in Tables 1-26. Generally, in a preferred embodiment, a test modulator is added to the cells prior to analysis. Moreover, screens are also provided to identify agents that modulate ovarian cancer, modulate ovarian cancer proteins, bind to an ovarian cancer protein, or interfere with the binding of an ovarian cancer protein and an antibody or other binding partner.

The term "test compound" or "drug candidate" or "modulator" or grammatical equivalents as used herein describes any molecule, e.g., protein, oligopeptide, small organic molecule, polysaccharide, polynucleotide, etc., to be tested for the capacity to directly or indirectly alter the ovarian cancer phenotype or the expression of an ovarian cancer sequence,

e.g., a nucleic acid or protein sequence. In preferred embodiments, modulators alter expression profiles, or expression profile nucleic acids or proteins provided herein. In one embodiment, the modulator suppresses an ovarian cancer phenotype, e.g., to a normal or non-malignant tissue fingerprint. In another embodiment, a modulator induced an ovarian cancer phenotype. Generally, a plurality of assay mixtures are run in parallel with different agent concentrations to obtain a differential response to the various concentrations. Typically, one of these concentrations serves as a negative control, e.g., at zero concentration or below the level of detection.

Drug candidates encompass numerous chemical classes, though typically they are organic molecules, preferably small organic compounds having a molecular weight of more than 100 and less than about 2,500 daltons. Preferred small molecules are less than 2000, or less than 1500 or less than 1000 or less than 500 D. Candidate agents comprise functional groups necessary for structural interaction with proteins, particularly hydrogen bonding, and typically include at least an amine, carbonyl, hydroxyl or carboxyl group, preferably at least two of the functional chemical groups. The candidate agents often comprise cyclical carbon or heterocyclic structures and/or aromatic or polyaromatic structures substituted with one or more of the above functional groups. Candidate agents are also found among biomolecules including peptides, saccharides, fatty acids, steroids, purines, pyrimidines, derivatives, structural analogs or combinations thereof. Particularly preferred are peptides.

In one aspect, a modulator will neutralize the effect of an ovarian cancer protein. By "neutralize" is meant that activity of a protein is inhibited or blocked and the consequent effect on the cell.

In certain embodiments, combinatorial libraries of potential modulators will be screened for an ability to bind to an ovarian cancer polypeptide or to modulate activity. Conventionally, new chemical entities with useful properties are generated by identifying a chemical compound (called a "lead compound") with some desirable property or activity, e.g., inhibiting activity, creating variants of the lead compound, and evaluating the property and activity of those variant compounds. Often, high throughput screening (HTS) methods are employed for such an analysis.

In one preferred embodiment, high throughput screening methods involve providing a library containing a large number of potential therapeutic compounds (candidate compounds). Such "combinatorial chemical libraries" are then screened in one or more assays to identify those library members (particular chemical species or subclasses) that

display a desired characteristic activity. The compounds thus identified can serve as conventional "lead compounds" or can themselves be used as potential or actual therapeutics.

A combinatorial chemical library is a collection of diverse chemical compounds generated by either chemical synthesis or biological synthesis by combining a number of chemical "building blocks" such as reagents. For example, a linear combinatorial chemical library, such as a polypeptide (e.g., muten) library, is formed by combining a set of chemical building blocks called amino acids in every possible way for a given compound length (e.g., the number of amino acids in a polypeptide compound). Millions of chemical compounds can be synthesized through such combinatorial mixing of chemical building blocks. See, e.g., Gallop, et al. (1994) J. Med. Chem. 37:1233-1251.

Preparation and screening of combinatorial chemical libraries is well known to those of skill in the art. Such combinatorial chemical libraries include, but are not limited to, peptide libraries (see, e.g., U.S. Patent No. 5,010,175; Furka (1991) Pept. Prot. Res. 37:487-493; and Houghton, et al. (1991) Nature 354:84-88), peptoids (PCT Publication No. WO 91/19735), encoded peptides (PCT Publication WO 93/20242), random bio-oligomers (PCT Publication WO 92/00091), benzodiazepines (U.S. Pat. No. 5,288,514), diversomers such as hydantoin, benzodiazepines and dipeptides (Hobbs, et al. (1993) Proc. Natl. Acad. Sci. USA 90:5909-913), vinylogous polypeptides (Hagihara, et al. (1992) J. Amer. Chem. Soc. 114:6568-570), non-peptidic peptidomimetics with a Beta-D-Glucose scaffolding.

(Hirschmann, et al. (1992) J. Amer. Chem. Soc. 114:9217-218), analogous organic syntheses of small compound libraries (Chen, et al. (1994) J. Amer. Chem. Soc. 116:2661-662), oligocarbamates (Cho, et al. (1993) Science 261:1303-305), and/or peptidyl phosphonates (Campbell, et al. (1994) J. Org. Chem. 59:658-xxx). See, generally, Gordon, et al. (1994) J. Med. Chem. 37:1385-401, nucleic acid libraries (see, e.g., Stratagene, Corp.), peptide nucleic acid libraries (see, e.g., U.S. Patent 5,539,083), antibody libraries (see, e.g., Vaughn, et al. (1996) Nature Biotechnology 14:309-314; and PCT/US96/10287), carbohydrate libraries (see, e.g., Liang, et al. (1996) Science 274:1520-1522; and U.S. Patent No. 5,593,853), and small organic molecule libraries (see, e.g., benzodiazepines, page 33, Baum (Jan. 18, 1993) C&E News; isoprenoids, U.S. Patent No. 5,569,588; thiazolidinones and metathiazanones, U.S. Patent No. 5,549,974; pyrrolidines, U.S. Patent Nos. 5,525,735 and 5,519,134; morpholino compounds, U.S. Patent No. 5,506,337; benzodiazepines, U.S. Patent No. 5,288,514; and the like).

Devices for the preparation of combinatorial libraries are commercially available.

See, e.g., 357 MPS, 390 MPS, Advanced Chem Tech, Louisville KY; Symphony, Rainin, Woburn, MA; 433A Applied Biosystems, Foster City, CA; 9050 Plus, Millipore, Bedford, MA.

A number of well known robotic systems have also been developed for solution phase chemistries. These systems include automated workstations like the automated synthesis apparatus developed by Takeda Chemical Industries, LTD. (Osaka, Japan) and many robotic systems utilizing robotic arms (Zymate II, Zymark Corporation, Hopkinton, MA; Orca, Hewlett-Packard, Palo Alto, CA), which mimic the manual synthetic operations performed by a chemist. Any of the above devices are suitable for use with the present invention. The nature and implementation of modifications to these devices (if any) so that they can operate as discussed herein will be apparent to persons skilled in the relevant art. In addition, numerous combinatorial libraries are themselves commercially available (see, e.g.,

ComGenex, Princeton, N.J.; Asinex, Moscow, RU; Tripos, Inc., St. Louis, MO; ChemStar, Ltd, Moscow, RU; 3D Pharmaceuticals, Exton, PA; Martek Biosciences, Columbia, MD; etc.).

The assays to identify modulators are amenable to high throughput screening.

Preferred assays thus detect enhancement or inhibition of ovarian cancer gene transcription, inhibition or enhancement of polypeptide expression, and inhibition or enhancement of polypeptide activity.

High throughput assays for the presence, absence, quantification, or other properties of particular nucleic acids or protein products are well known to those of skill in the art. Similarly, binding assays and reporter gene assays are similarly well known. Thus, e.g., U.S. Patent No. 5,559,410 discloses high throughput screening methods for proteins, U.S. Patent No. 5,585,639 discloses high throughput screening methods for nucleic acid binding (e.g., in arrays), while U.S. Patent Nos. 5,576,220 and 5,541,061 disclose high throughput methods of screening for ligand/antibody binding.

In addition, high throughput screening systems are commercially available (see, e.g., Zymark Corp., Hopkinton, MA; Air Technical Industries, Mentor, OH; Beckman Instruments, Inc. Fullerton, CA; Precision Systems, Inc., Natick, MA, etc.). These systems typically automate entire procedures, including all sample and reagent pipetting, liquid dispensing, timed incubations, and final readings of the microplate in detector(s) appropriate for the assay. These configurable systems provide high throughput and rapid start up as well as a high degree of flexibility and customization. The manufacturers of such systems provide

detailed protocols for various high throughput systems. Thus, e.g., Zymark Corp. provides technical bulletins describing screening systems for detecting the modulation of gene transcription, ligand binding, and the like.

In one embodiment, modulators are proteins, often naturally occurring proteins or fragments of naturally occurring proteins. Thus, e.g., cellular extracts containing proteins, or random or directed digests of proteinaceous cellular extracts, may be used. In this way libraries of proteins may be made for screening in the methods of the invention. Particularly preferred in this embodiment are libraries of bacterial, fungal, viral, and mammalian proteins, with the latter being preferred, and human proteins being especially preferred. Particularly useful test compound will be directed to the class of proteins to which the target belongs, e.g., substrates for enzymes or ligands and receptors.

In a preferred embodiment, modulators are peptides of from about 5 to about 30 amino acids, with from about 5 to about 20 amino acids being preferred, and from about 7 to about 15 being particularly preferred. The peptides may be digests of naturally occurring proteins as is outlined above, random peptides, or "biased" random peptides. By "randomized" or grammatical equivalents herein is meant that each nucleic acid and peptide consists of essentially random nucleotides and amino acids, respectively. Since generally these random peptides (or nucleic acids, discussed below) are chemically synthesized, they may incorporate any nucleotide or amino acid at any position. The synthetic process can be designed to generate randomized proteins or nucleic acids, to allow the formation of all or most of the possible combinations over the length of the sequence, thus forming a library of randomized candidate bioactive proteinaceous agents.

In one embodiment, the library is fully randomized, with no sequence preferences or constants at any position. In a preferred embodiment, the library is biased. That is, some positions within the sequence are either held constant, or are selected from a limited number of possibilities. For example, in a preferred embodiment, the nucleotides or amino acid residues are randomized within a defined class, e.g., of hydrophobic amino acids, hydrophilic residues, sterically biased (either small or large) residues, towards the creation of nucleic acid binding domains, the creation of cysteines, for cross-linking, prolines for SH-3 domains, serines, threonines, tyrosines or histidines for phosphorylation sites, etc., or to purines, etc.

Modulators of ovarian cancer can also be nucleic acids, as defined above.

As described above generally for proteins, nucleic acid modulating agents may be naturally occurring nucleic acids, random nucleic acids, or "biased" random nucleic acids.

For example, digests of procaryotic or eucaryotic genomes may be used as is outlined above for proteins.

In a preferred embodiment, the candidate compounds are organic chemical moieties, a wide variety of which are available in the literature.

After the candidate agent has been added and the cells allowed to incubate for some period of time, the sample containing a target sequence to be analyzed is added to the biochip. If required, the target sequence is prepared using known techniques. For example, the sample may be treated to lyse the cells, using known lysis buffers, electroporation, etc., with purification and/or amplification such as PCR, performed as appropriate. For example, an in vitro transcription with labels covalently attached to the nucleotides is performed. Generally, the nucleic acids are labeled with biotin-FTTC or PE, or with cy3 or cy5.

In a preferred embodiment, the target sequence is labeled with, e.g., a fluorescent, a chemiluminescent, a chemical, or a radioactive signal, to provide a means of detecting the target sequence's specific binding to a probe. The label also can be an enzyme, such as, alkaline phosphatase or horseradish peroxidase, which when provided with an appropriate substrate produces a product that can be detected. Alternatively, the label can be a labeled compound or small molecule, such as an enzyme inhibitor, that binds but is not catalyzed or altered by the enzyme. The label also can be a moiety or compound, such as, an epitope tag or biotin which specifically binds to streptavidin. For the example of biotin, the streptavidin is labeled as described above, thereby, providing a detectable signal for the bound target sequence. Unbound labeled streptavidin is typically removed prior to analysis.

As will be appreciated by those in the art, these assays can be direct hybridization assays or can comprise "sandwich assays", which include the use of multiple probes, as is generally outlined in U.S. Patent Nos. 5,681,702; 5,597,909; 5,545,730; 5,594,117; 5,591,584; 5,571,670; 5,580,731; 5,571,670; 5,591,584; 5,624,802; 5,635,352; 5,594,118; 5,359,100; 5,124,246; and 5,681,697, each of which is hereby incorporated by reference. In this embodiment, in general, the target nucleic acid is prepared as outlined above, and then added to the biochip comprising a plurality of nucleic acid probes, under conditions that allow the formation of a hybridization complex.

A variety of hybridization conditions may be used in the present invention, including high, moderate and low stringency conditions as outlined above. The assays are generally run under stringency conditions which allows formation of the label probe hybridization complex only in the presence of target. Stringency can be controlled by altering a step

parameter that is a thermodynamic variable, including, but not limited to, temperature, formamide concentration, salt concentration, chaotropic salt concentration pH, organic solvent concentration, etc.

These parameters may also be used to control non-specific binding, as is generally outlined in U.S. Patent No. 5,681,697. Thus it may be desirable to perform certain steps at higher stringency conditions to reduce non-specific binding.

The reactions outlined herein may be accomplished in a variety of ways. Components of the reaction may be added simultaneously, or sequentially, in different orders, with preferred embodiments outlined below. In addition, the reaction may include a variety of other reagents. These include salts, buffers, neutral proteins, e.g., albumin, detergents, etc. which may be used to facilitate optimal hybridization and detection, and/or reduce non-specific or background interactions. Reagents that otherwise improve the efficiency of the assay, such as protease inhibitors, nuclease inhibitors, anti-microbial agents, etc., may also be used as appropriate, depending on the sample preparation methods and purity of the target.

The assay data are analyzed to determine the expression levels, and changes in expression levels as between states, of individual genes, forming a gene expression profile.

Screens are performed to identify modulators of the ovarian cancer phenotype. In one embodiment, screening is performed to identify modulators that can induce or suppress a particular expression profile, thus preferably generating the associated phenotype. In another embodiment, e.g., for diagnostic applications, having identified differentially expressed genes important in a particular state, screens can be performed to identify modulators that alter expression of individual genes. In another embodiment, screening is performed to identify modulators that alter a biological function of the expression product of a differentially expressed gene. Again, having identified the importance of a gene in a particular state, screens are performed to identify agents that bind and/or modulate the biological activity of the gene product.

In addition screens can be done for genes that are induced in response to a candidate agent. After identifying a modulator based upon its ability to suppress an ovarian cancer expression pattern leading to a normal expression pattern, or to modulate a single ovarian cancer gene expression profile so as to mimic the expression of the gene from normal tissue, a screen as described above can be performed to identify genes that are specifically modulated in response to the agent. Comparing expression profiles between normal tissue and agent treated ovarian cancer tissue reveals genes that are not expressed in normal tissue

or ovarian cancer tissue, but are expressed in agent treated tissue. These agent-specific sequences can be identified and used by methods described herein for ovarian cancer genes or proteins. In particular these sequences and the proteins they encode find use in marking or identifying agent treated cells. In addition, antibodies can be raised against the agent induced proteins and used to target novel therapeutics to the treated ovarian cancer tissue sample.

Thus, in one embodiment, a test compound is administered to a population of ovarian cancer cells, that have an associated ovarian cancer expression profile. By "administration" or "contacting" herein is meant that the candidate agent is added to the cells in such a manner as to allow the agent to act upon the cell, whether by uptake and intracellular action, or by action at the cell surface. In some embodiments, nucleic acid encoding a proteinaceous candidate agent (e.g., a peptide) may be put into a viral construct such as an adenoviral or retroviral construct, and added to the cell, such that expression of the peptide agent is accomplished, e.g., PCT US97/01019. Regulatable gene therapy systems can also be used.

Once the test compound has been administered to the cells, the cells can be washed if desired and are allowed to incubate under preferably physiological conditions for some period of time. The cells are then harvested and a new gene expression profile is generated, as outlined herein.

Thus, e.g., ovarian cancer or non-malignant tissue may be screened for agents that modulate, e.g., induce or suppress the ovarian cancer phenotype. A change in at least one gene, preferably many, of the expression profile indicates that the agent has an effect on ovarian cancer activity. By defining such a signature for the ovarian cancer phenotype, screens for new drugs that alter the phenotype can be devised. With this approach, the drug target need not be known and need not be represented in the original expression screening platform, nor does the level of transcript for the target protein need to change.

In a preferred embodiment, as outlined above, screens may be done on individual genes and gene products (proteins). That is, having identified a particular differentially expressed gene as important in a particular state, screening of modulators of either the expression of the gene or the gene product itself can be done. The gene products of differentially expressed genes are sometimes referred to herein as "ovarian cancer proteins" or a "ovarian cancer modulatory protein". The ovarian cancer modulatory protein may be a fragment, or alternatively, be the full length protein to the fragment encoded by the nucleic acids of the Tables. Preferably, the ovarian cancer modulatory protein is a fragment. In a preferred embodiment, the ovarian cancer amino acid sequence which is used to determine

sequence identity or similarity is encoded by a nucleic acid of the Tables. In another embodiment, the sequences are naturally occurring allelic variants of a protein encoded by a nucleic acid of the Tables. In another embodiment, the sequences are sequence variants as further described herein.

Preferably, the ovarian cancer modulatory protein is a fragment of approximately 14 to 24 amino acids long. More preferably the fragment is a soluble fragment. Preferably, the fragment includes a non-transmembrane region. In a preferred embodiment, the fragment has an N-terminal Cys to aid in solubility. In another embodiment, the C-terminus of the fragment is kept as a free acid and the N-terminus is a free amine to aid in coupling, e.g., to cysteine. Or, the ovarian cancer proteins are conjugated to an immunogenic agent, e.g., to BSA.

Measurements of ovarian cancer polypeptide activity, or of ovarian cancer or the ovarian cancer phenotype can be performed using a variety of assays. For example, the effects of the test compounds upon the function of the ovarian cancer polypeptides can be measured by examining parameters described above. A suitable physiological change that affects activity can be used to assess the influence of a test compound on the polypeptides of this invention. When the functional consequences are determined using intact cells or animals, one can also measure a variety of effects such as, in the case of ovarian cancer associated with tumors, tumor growth, tumor metastasis, neovascularization, hormone release, transcriptional changes to both known and uncharacterized genetic markers (e.g., northern blots), changes in cell metabolism such as cell growth or pH changes, and changes in intracellular second messengers such as cGMP. In the assays of the invention, mammalian ovarian cancer polypeptide is typically used, e.g., mouse, preferably human.

Assays to identify compounds with modulating activity can be performed in vitro. For example, an ovarian cancer polypeptide is first contacted with a potential modulator and incubated for a suitable amount of time, e.g., from 0.5 to 48 hours. In one embodiment, the ovarian cancer polypeptide levels are determined in vitro by measuring the level of protein or mRNA. The level of protein is measured using immunoassays such as western blotting, ELISA and the like with an antibody that selectively binds to the ovarian cancer polypeptide or a fragment thereof. For measurement of mRNA, amplification, e.g., using PCR, LCR, or hybridization assays, e.g., northern hybridization, RNase protection, dot blotting, are preferred. The level of protein or mRNA is detected using directly or indirectly labeled detection agents, e.g., fluorescently or radioactively labeled nucleic acids, radioactively or

enzymatically labeled antibodies, and the like, as described herein.

Alternatively, a reporter gene system can be devised using the ovarian cancer protein promoter operably linked to a reporter gene such as luciferase, green fluorescent protein, CAT, or β -gal. The reporter construct is typically transfected into a cell. After treatment with a potential modulator, the amount of reporter gene transcription, translation, or activity is measured according to standard techniques known to those of skill in the art.

In a preferred embodiment, as outlined above, screens may be done on individual genes and gene products (proteins). That is, having identified a particular differentially expressed gene as important in a particular state, screening of modulators of the expression of the gene or the gene product itself can be done. The gene products of differentially expressed genes are sometimes referred to herein as "ovarian cancer proteins." The ovarian cancer protein may be a fragment, or alternatively, be the full length protein to a fragment shown herein.

In one embodiment, screening for modulators of expression of specific genes is performed. Typically, the expression of only one or a few genes are evaluated. In another embodiment, screens are designed to first find compounds that bind to differentially expressed proteins. These compounds are then evaluated for the ability to modulate differentially expressed activity. Moreover, once initial candidate compounds are identified, variants can be further screened to better evaluate structure activity relationships.

In a preferred embodiment, binding assays are done. In general, purified or isolated gene product is used; that is, the gene products of one or more differentially expressed nucleic acids are made. For example, antibodies are generated to the protein gene products, and standard immunoassays are run to determine the amount of protein present. Alternatively, cells comprising the ovarian cancer proteins can be used in the assays.

Thus, in a preferred embodiment, the methods comprise combining an ovarian cancer protein and a candidate compound, and determining the binding of the compound to the ovarian cancer protein. Preferred embodiments utilize the human ovarian cancer protein, although other mammalian proteins, e.g., counterparts, may also be used, e.g., for the development of animal models of human disease. In some embodiments, as outlined herein, variant or derivative ovarian cancer proteins may be used.

Generally, in a preferred embodiment of the methods herein, the ovarian cancer protein or the candidate agent is non-diffusably bound to an insoluble support having isolated sample receiving areas (e.g., a microtiter plate, an array, etc.). The insoluble supports may be

made of any composition to which the compositions can be bound, is readily separated from soluble material, and is otherwise compatible with the overall method of screening. The surface of such supports may be solid or porous and of any convenient shape. Examples of suitable insoluble supports include microtiter plates, arrays, membranes and beads. These are typically made of glass, plastic (e.g., polystyrene), polysaccharides, nylon or nitrocellulose, teflon™, etc. Microtiter plates and arrays are especially convenient because a large number of assays can be carried out simultaneously, using small amounts of reagents and samples.

The particular manner of binding of the composition is not crucial so long as it is compatible with the reagents and overall methods of the invention, maintains the activity of the composition and is non-diffusible. Preferred methods of binding include the use of antibodies (which do not sterically block either the ligand binding site or activation sequence when the protein is bound to the support), direct binding to "sticky" or ionic supports, chemical crosslinking, the synthesis of the protein or agent on the surface, etc. Following binding of the protein or agent, excess unbound material is removed by washing. The sample receiving areas may then be blocked through incubation with bovine serum albumin (BSA), casein or other innocuous protein or other moiety.

In a preferred embodiment, the ovarian cancer protein is bound to the support, and a test compound is added to the assay. Alternatively, the candidate agent is bound to the support and the ovarian cancer protein is added. Novel binding agents include specific antibodies, non-natural binding agents identified in screens of chemical libraries, peptide analogs, etc. Of particular interest are screening assays for agents that have a low toxicity for human cells. A wide variety of assays may be used for this purpose, including labeled in vitro protein-protein binding assays, electrophoretic mobility shift assays, immunoassays for protein binding, functional assays (phosphorylation assays, etc.) and the like.

The determination of the binding of the test modulating compound to the ovarian cancer protein may be done in a number of ways. In a preferred embodiment, the compound is labeled, and binding determined directly, e.g., by attaching all or a portion of the ovarian cancer protein to a solid support, adding a labeled candidate agent (e.g., a fluorescent label), washing off excess reagent, and determining whether the label is present on the solid support. Various blocking and washing steps may be utilized as appropriate.

In some embodiments, only one of the components is labeled, e.g., the proteins (or proteinaceous candidate compounds) can be labeled. Alternatively, more than one

component can be labeled with different labels, e.g., 125I for the proteins and a fluorophore for the compound. Proximity reagents, e.g., quenching or energy transfer reagents are also useful.

In one embodiment, the binding of the test compound is determined by competitive binding assay. The competitor is a binding moiety known to bind to the target molecule (e.g., an ovarian cancer protein), such as an antibody, peptide, binding partner, ligand, etc. Under certain circumstances, there may be competitive binding between the compound and the binding moiety, with the binding moiety displacing the compound. In one embodiment, the test compound is labeled. Either the compound, or the competitor, or both, is added first to the protein for a time sufficient to allow binding, if present. Incubations may be performed at a temperature which facilitates optimal activity, typically 4-40° C. Incubation periods are typically optimized, e.g., to facilitate rapid high throughput screening. Typically between 0.1 and 1 hr will be sufficient. Excess reagent is generally removed or washed away. The second component is then added, and the presence or absence of the labeled component is followed, to indicate binding.

In a preferred embodiment, the competitor is added first, followed by the test compound. Displacement of the competitor is an indication that the test compound is binding to the ovarian cancer protein and thus is capable of binding to, and potentially modulating, the activity of the ovarian cancer protein. In this embodiment, either component can be labeled. Thus, e.g., if the competitor is labeled, the presence of label in the wash solution indicates displacement by the agent. Alternatively, if the test compound is labeled, the presence of the label on the support indicates displacement.

In an alternative embodiment, the test compound is added first, with incubation and washing, followed by the competitor. The absence of binding by the competitor may indicate that the test compound is bound to the ovarian cancer protein with a higher affinity. Thus, if the test compound is labeled, the presence of the label on the support, coupled with a lack of competitor binding, may indicate that the test compound is capable of binding to the ovarian cancer protein.

In a preferred embodiment, the methods comprise differential screening to identify agents that are capable of modulating the activity of the ovarian cancer proteins. In this embodiment, the methods comprise combining an ovarian cancer protein and a competitor in a first sample. A second sample comprises a test compound, an ovarian cancer protein, and a

competitor. The binding of the competitor is determined for both samples, and a change, or difference in binding between the two samples indicates the presence of an agent capable of binding to the ovarian cancer protein and potentially modulating its activity. That is, if the binding of the competitor is different in the second sample relative to the first sample, the agent is capable of binding to the ovarian cancer protein.

Alternatively, differential screening is used to identify drug candidates that bind to the native ovarian cancer protein, but cannot bind to modified ovarian cancer proteins. The structure of the ovarian cancer protein may be modeled, and used in rational drug design to synthesize agents that interact with that site. Drug candidates that affect the activity of an ovarian cancer protein are also identified by screening drugs for the ability to either enhance or reduce the activity of the protein.

Positive controls and negative controls may be used in the assays. Preferably control and test samples are performed in at least triplicate to obtain statistically significant results. Incubation of all samples is for a time sufficient for the binding of the agent to the protein. Following incubation, samples are washed free of non-specifically bound material and the amount of bound, generally labeled agent determined. For example, where a radiolabel is employed, the samples may be counted in a scintillation counter to determine the amount of bound compound.

A variety of other reagents may be included in the screening assays. These include reagents like salts, neutral proteins, e.g., albumin, detergents, etc. which may be used to facilitate optimal protein-protein binding and/or reduce non-specific or background interactions. Also reagents that otherwise improve the efficiency of the assay, such as protease inhibitors, nuclease inhibitors, anti-microbial agents, etc., may be used. The mixture of components may be added in an order that provides for the requisite binding.

In a preferred embodiment, the invention provides methods for screening for a compound capable of modulating the activity of an ovarian cancer protein. The methods comprise adding a test compound, as defined above, to a cell comprising ovarian cancer proteins. Preferred cell types include almost any cell. The cells contain a recombinant nucleic acid that encodes an ovarian cancer protein. In a preferred embodiment, a library of candidate agents are tested on a plurality of cells.

In one aspect, the assays are evaluated in the presence or absence or previous or subsequent exposure of physiological signals, e.g., hormones, antibodies, peptides, antigens, cytokines, growth factors, action potentials, pharmacological agents including

chemotherapeutics, radiation, carcinogenics, or other cells (e.g., cell-cell contacts). In another example, the determinations are determined at different stages of the cell cycle process.

In this way, compounds that modulate ovarian cancer agents are identified. Compounds with pharmacological activity are able to enhance or interfere with the activity of the ovarian cancer protein. Once identified, similar structures are evaluated to identify critical structural feature of the compound.

In one embodiment, a method of inhibiting ovarian cancer cell division is provided. The method comprises administration of an ovarian cancer inhibitor. In another embodiment, a method of inhibiting ovarian cancer is provided. The method comprises administration of an ovarian cancer inhibitor. In a further embodiment, methods of treating cells or individuals with ovarian cancer are provided. The method comprises administration of an ovarian cancer inhibitor.

In one embodiment, an ovarian cancer inhibitor is an antibody as discussed above. In another embodiment, the ovarian cancer inhibitor is an antisense or RNAi molecule.

A variety of cell viability, growth, proliferation, and metastasis assays are known to those of skill in the art, as described below.

Soft agar growth or colony formation in suspension

Normal cells require a solid substrate to attach and grow. When the cells are transformed, they lose this phenotype and grow detached from the substrate. For example, transformed cells can grow in stirred suspension culture or suspended in semi-solid media, such as semi-solid or soft agar. The transformed cells, when transfected with tumor suppressor genes, regenerate normal phenotype and require a solid substrate to attach and grow. Soft agar growth or colony formation in suspension assays can be used to identify modulators of ovarian cancer sequences, which when expressed in host cells, inhibit abnormal cellular proliferation and transformation. A therapeutic compound would reduce or eliminate the host cells' ability to grow in stirred suspension culture or suspended in semi-solid media, such as semi-solid or soft.

Techniques for soft agar growth or colony formation in suspension assays are described in Freshney (1994) *Culture of Animal Cells: A Manual of Basic Technique* (3d ed.) Wiley-Liss, herein incorporated by reference. See also, the methods section of Garkavtsev, et al. (1996), supra, herein incorporated by reference.

Contact inhibition and density limitation of growth

Normal cells typically grow in a flat and organized pattern in a petri dish until they touch other cells. When the cells touch one another, they are contact inhibited and stop growing. When cells are transformed, however, the cells are not contact inhibited and continue to grow to high densities in disorganized foci. Thus, the transformed cells grow to a higher saturation density than normal cells. This can be detected morphologically by the formation of a disoriented monolayer of cells or rounded cells in foci within the regular pattern of normal surrounding cells. Alternatively, labeling index with (^3H)-thymidine at saturation density can be used to measure density limitation of growth. See, e.g., Freshney (1994), supra. The transformed cells, when transfected with tumor suppressor genes, regenerate a normal phenotype and become contact inhibited and would grow to a lower density.

In this assay, labeling index with (^3H)-thymidine at saturation density is a preferred method of measuring density limitation of growth. Transformed host cells are transfected with an ovarian cancer-associated sequence and are grown for 24 hr at saturation density in non-limiting medium conditions. The percentage of cells labeling with (^3H)-thymidine is determined autoradiographically. See, e.g., Freshney (1994), supra.

Growth factor or serum dependence

Transformed cells typically have a lower serum dependence than their normal counterparts. See, e.g., Temin (1966) *J. Nat'l. Cancer Inst.* 37:167-175; Eagle, et al. (1970) *J. Exp. Med.* 131:836-879; and Freshney, supra. This is in part due to release of various growth factors by the transformed cells. Growth factor or serum dependence of transformed host cells can be compared with that of control.

Tumor specific markers levels

Tumor cells release an increased amount of certain factors (hereinafter "tumor specific markers") than their normal counterparts. For example, plasminogen activator (PA) is released from human glioma at a higher level than from normal brain cells (see, e.g., Gullino, pp. 178-184 "Angiogenesis, tumor vascularization, and potential interference with tumor growth" in Mihich (ed. 1985) *Biological Responses in Cancer* Plenum. Similarly,

tumor angiogenesis factor (TAF) is released at a higher level in tumor cells than their normal counterparts. See, e.g., Folkman (1992) Sem Cancer Biol. 3:89-96.

Various techniques which measure the release of these factors are described in

5 Freshney (1994), supra. Also, see, Unkeless, et al. (1974) J. Biol. Chem. 249:4295-4305; Strickland and Beers (1976) J. Biol. Chem. 251:5694-5702; Whur, et al. (1980) Br. J. Cancer 42:305-312; Gullino, pp. 178-184 "Angiogenesis, tumor vascularization, and potential interference with tumor growth" in Milich (ed. 1985) Biological Responses in Cancer Plenum; and Freshney (1985) Anticancer Res. 5:111-130.

10 Invasiveness into Matrigel

The degree of invasiveness into Matrigel or some other extracellular matrix constituent can be used as an assay to identify compounds that modulate ovarian cancer-associated sequences. Tumor cells exhibit a good correlation between malignancy and invasiveness of cells into Matrigel or some other extracellular matrix constituent. In this

15 assay, tumorigenic cells are typically used as host cells. Expression of a tumor suppressor gene in these host cells would decrease invasiveness of the host cells.

Alternatively, the level of invasion of host cells can be measured by using filters coated with Matrigel or some other extracellular matrix constituent. Penetration into the gel, or through to the distal side of the filter, is rated as invasiveness, and rated histologically by

20 number of cells and distance moved, or by pre-labeling the cells with ¹²⁵I and counting the radioactivity on the distal side of the filter or bottom of the dish. See, e.g., Freshney (1984), supra.

Tumor growth in vivo

25 Effects of ovarian cancer-associated sequences on cell growth can be tested in transgenic or immune-suppressed mice. Knock-out transgenic mice can be made, in which the ovarian cancer gene is disrupted or in which an ovarian cancer gene is inserted. Knock-out transgenic mice can be made by insertion of a marker gene or other heterologous gene into the endogenous ovarian cancer gene site in the mouse genome via homologous recombination. Such mice can also be made by substituting the endogenous ovarian cancer gene with a mutated version of the ovarian cancer gene, or by mutating the endogenous ovarian cancer gene, e.g., by exposure to carcinogens.

A DNA construct is introduced into the nuclei of embryonic stem cells. Cells containing the newly engineered genetic lesion are injected into a host mouse embryo, which is re-implanted into a recipient female. Some of these embryos develop into chimeric mice that possess germ cells partially derived from the mutant cell line. By breeding the chimeric mice it is possible to obtain a new line of mice containing the introduced genetic lesion. See, e.g., Capechi, et al. (1989) Science 244:1288-1292. Chimeric targeted mice can be derived according to Hogan, et al. (1988) Manipulating the Mouse Embryo: A Laboratory Manual CSH Press; and Robertson (ed. 1987) Teratocarcinomas and Embryonic Stem Cells: A Practical Approach IRL Press, Washington, D.C.

10 Alternatively, various immune-suppressed or immune-deficient host animals can be used. For example, genetically athymic "nude" mouse (see, e.g., Giovanella, et al. (1974) J. Nat'l Cancer Inst. 52:921-930), a SCID mouse, a thymectomized mouse, or an irradiated mouse (see, e.g., Bradley, et al. (1978) Br. J. Cancer 38:263-272; Selby, et al. (1980) Br. J. Cancer 41:52-61) can be used as a host. Transplantable tumor cells (typically about 10⁶ cells) injected into isogenic hosts will produce invasive tumors in a high proportions of cases, while normal cells of similar origin will not. In hosts which developed invasive tumors, cells expressing an ovarian cancer-associated sequences are injected subcutaneously. After a suitable length of time, preferably 4-8 weeks, tumor growth is measured (e.g., by volume or by its two largest dimensions) and compared to the control. Tumors that have statistically

20 significant reduction (using, e.g., Student's T test) are said to have inhibited growth.

Polynucleotide modulators of ovarian cancer

Antisense and RNAi Polynucleotides

25 In certain embodiments, the activity of an ovarian cancer-associated protein is down-regulated, or entirely inhibited, by the use of antisense polynucleotide, e.g., a nucleic acid complementary to, and which can preferably hybridize specifically to, a coding mRNA nucleic acid sequence, e.g., an ovarian cancer protein mRNA, or a subsequence thereof. Binding of the antisense polynucleotide to the mRNA reduces the translation and/or stability of the mRNA.

30 In the context of this invention, antisense polynucleotides can comprise naturally-occurring nucleotides, or synthetic species formed from naturally-occurring subunits or their close homologs. Antisense polynucleotides may also have altered sugar moieties or inter-

sugar linkages. Exemplary among these are the phosphorothioate and other sulfur containing species which are known for use in the art. Analogs are comprehended by this invention so long as they function effectively to hybridize with the ovarian cancer protein mRNA. See, e.g., Isis Pharmaceuticals, Carlsbad, CA; Sequitor, Inc., Natick, MA.

5 Such antisense polynucleotides can readily be synthesized using recombinant means, or can be synthesized in vitro. Equipment for such synthesis is sold by several vendors, including Applied Biosystems. The preparation of other oligonucleotides such as phosphorothioates and alkylated derivatives is also well known to those of skill in the art.

Antisense molecules as used herein include antisense or sense oligonucleotides.

10 Sense oligonucleotides can, e.g., be employed to block transcription by binding to the anti-sense strand. The antisense and sense oligonucleotide comprise a single-stranded nucleic acid sequence (either RNA or DNA) capable of binding to target mRNA (sense) or DNA (antisense) sequences for ovarian cancer molecules. A preferred antisense molecule is for an ovarian cancer sequences in Tables 1-26, or for a ligand or activator thereof. Antisense or sense oligonucleotides, according to the present invention, comprise a fragment generally at least about 14 nucleotides, preferably from about 14 to 30 nucleotides. An antisense or a sense oligonucleotide can be developed based upon a cDNA sequence encoding a given protein. See, e.g., Stein and Cohen (1988) Cancer Res. 48:2659-2668; and van der Kroel, et al. (1988) BioTechniques 6:958-976.

20 RNA interference is a mechanism to suppress gene expression in a sequence specific manner. See, e.g., Brumelkamp, et al. (2002) Scienceexpress (21March2002); Sharp (1999) Genes Dev. 13:139-141; and Cathew (2001) Curr. Op. Cell Biol. 13:244-248. In mammalian cells, short, e.g., 21 nt, double stranded small interfering RNAs (siRNA) have been shown to be effective at inducing an RNAi response. See, e.g., Elbashir, et al. (2001) Nature 411:494-498. The mechanism may be used to down-regulate expression levels of identified genes, e.g., treatment of or validation of relevance to disease.

Ribozymes

30 In addition to antisense polynucleotides, ribozymes can be used to target and inhibit transcription of ovarian cancer-associated nucleotide sequences. A ribozyme is an RNA molecule that catalytically cleaves other RNA molecules. Different kinds of ribozymes have been described, including group I ribozymes, hammerhead ribozymes, hairpin ribozymes, RNase P, and axthead ribozymes (see, e.g., Castanotto, et al. (1994) Adv. Pharmacol. 25: 289-

317 for a general review of the properties of different ribozymes).

The general features of hairpin ribozymes are described, e.g., in Hampel, et al. (1990) Nucl. Acids Res. 18:299-304; European Patent Publication No. 0 360 257; U.S. Patent No. 5,254,678. Methods of preparing them are well known to those of skill in the art. See, e.g., WO 94/26877; Ojwang, et al. (1993) Proc. Nat'l Acad. Sci. USA 90:6340-6344; Yamada, et al. (1994) Hum. Gene Ther. 1:39-45; Leavitt, et al. (1995) Proc. Nat'l Acad. Sci. USA 92:699-703; Leavitt, et al. (1994) Hum. Gene Ther. 5:1151-120; and Yamada, et al. (1994) Virology 205:121-126.

Polynucleotide modulators of ovarian cancer may be introduced into a cell containing the target nucleotide sequence by formation of a conjugate with a ligand binding molecule, as described in WO 91/04753. Suitable ligand binding molecules include, but are not limited to, cell surface receptors, growth factors, other cytokines, or other ligands that bind to cell surface receptors. Preferably, conjugation of the ligand binding molecule does not substantially interfere with the ability of the ligand binding molecule to bind to its corresponding molecule or receptor, or block entry of the sense or antisense oligonucleotide or its conjugated version into the cell. Alternatively, a polynucleotide modulator of ovarian cancer may be introduced into a cell containing the target nucleic acid sequence, e.g., by formation of an polynucleotide-lipid complex, as described in WO 90/10448. It is understood that the use of antisense molecules or knock out and knock in models may also be used in screening assays as discussed above, in addition to methods of treatment.

20 Thus, in one embodiment, methods of modulating ovarian cancer in cells or organisms are provided. In one embodiment, the methods comprise administering to a cell an anti-ovarian cancer antibody that reduces or eliminates the biological activity of an endogenous ovarian cancer protein. Alternatively, the methods comprise administering to a cell or organism a recombinant nucleic acid encoding an ovarian cancer protein. This may be accomplished in any number of ways. In a preferred embodiment, e.g., when the ovarian cancer sequence is down-regulated in ovarian cancer, such state may be reversed by increasing the amount of ovarian cancer gene product in the cell. This can be accomplished, e.g., by over-expressing the endogenous ovarian cancer gene or administering a gene encoding the ovarian cancer sequence, using known gene-therapy techniques, e.g., in a preferred embodiment, the gene therapy techniques include the incorporation of the exogenous gene using enhanced homologous recombination (EHR), e.g., as described in PCT/US93/03868, hereby incorporated by reference in its entirety. Alternatively, e.g., when

the ovarian cancer sequence is up-regulated in ovarian cancer, the activity of the endogenous ovarian cancer gene is decreased, e.g., by the administration of an ovarian cancer antisense or RNAi nucleic acid.

In one embodiment, the ovarian cancer proteins of the present invention may be used to generate polyclonal and monoclonal antibodies to ovarian cancer proteins. Similarly, the ovarian cancer proteins can be coupled, using standard technology, to affinity chromatography columns. These columns may then be used to purify ovarian cancer antibodies useful for production, diagnostic, or therapeutic purposes. In a preferred embodiment, the antibodies are generated to epitopes unique to an ovarian cancer protein; that is, the antibodies show little or no cross-reactivity to other proteins. The ovarian cancer antibodies may be coupled to standard affinity chromatography columns and used to purify ovarian cancer proteins. The antibodies may also be used as blocking polypeptides, as outlined above, since they will specifically bind to the ovarian cancer protein.

15 Methods of identifying variant ovarian cancer-associated sequences

Without being bound by theory, expression of various ovarian cancer sequences is correlated with ovarian cancer. Accordingly, disorders based on mutant or variant ovarian cancer genes may be determined. In one embodiment, the invention provides methods for identifying cells containing variant ovarian cancer genes, e.g., determining all or part of the sequence of at least one endogenous ovarian cancer genes in a cell. This may be accomplished using any number of sequencing techniques. In a preferred embodiment, the invention provides methods of identifying the ovarian cancer genotype of an individual, e.g., determining all or part of the sequence of at least one ovarian cancer gene of the individual. This is generally done in at least one tissue of the individual, and may include the evaluation of a number of tissues or different samples of the same tissue. The method may include comparing the sequence of the sequenced ovarian cancer gene to a known ovarian cancer gene, e.g., a wild-type gene.

The sequence of all or part of the ovarian cancer gene can then be compared to the sequence of a known ovarian cancer gene to determine if any differences exist. This can be done using any number of known homology programs, such as Bestfit, etc. In a preferred embodiment, the presence of a difference in the sequence between the ovarian cancer gene of the patient and the known ovarian cancer gene correlates with a disease state or a propensity for a disease state, as outlined herein.

In a preferred embodiment, the ovarian cancer genes are used as probes to determine the number of copies of the ovarian cancer gene in the genome.

In another preferred embodiment, the ovarian cancer genes are used as probes to determine the chromosomal localization of the ovarian cancer genes. Information such as chromosomal localization finds use in providing a diagnosis or prognosis in particular when chromosomal abnormalities such as translocations, and the like are identified in the ovarian cancer gene locus.

Administration of pharmaceutical and vaccine compositions

In one embodiment, a therapeutically effective dose of an ovarian cancer protein or modulator thereof, is administered to a patient. By "therapeutically effective dose" herein is meant a dose that produces effects for which it is administered. The exact dose will depend on the purpose of the treatment, and will be ascertainable by one skilled in the art using known techniques. See, e.g., Ansel, et al. (1999) Pharmaceutical Dosage Forms and Drug Delivery Systems Lippincott; Lieberman (1992) Pharmaceutical Dosage Forms (vols. 1-3) Dekker, ISBN 0824770846, 082476918X, 0824712692, 0824716981; Lloyd (1999) The Art, Science and Technology of Pharmaceutical Compounding Amer. Pharmaceutical Assn.; and Pickar (1999) Dosage Calculations Thomson. Adjustments for ovarian cancer degradation, systemic versus localized delivery, and rate of new protease synthesis, as well as the age, body weight, general health, sex, diet, time of administration, drug interaction, and the severity of the condition may be necessary, and will be ascertainable with routine experimentation by those skilled in the art. U.S. Patent Application No. 09/687,576, further discloses the use of compositions and methods of diagnosis and treatment in ovarian cancer is hereby expressly incorporated by reference.

A "patient" for the purposes of the present invention includes both humans and other animals, particularly mammals. Thus the methods are applicable to both human therapy and veterinary applications. In the preferred embodiment the patient is a mammal, preferably a primate, and in the most preferred embodiment the patient is human.

The administration of the ovarian cancer proteins and modulators thereof of the present invention can be done in a variety of ways as discussed above, including, but not limited to, orally, subcutaneously, intravenously, intra-nasally, transdermally, intraperitoneally, intramuscularly, intrapulmonary, vaginally, rectally, or intraocularly. In some instances, e.g., in the treatment of wounds and inflammation, the ovarian cancer

proteins and modulators may be directly applied as a solution or spray.

The pharmaceutical compositions of the present invention comprise an ovarian cancer protein in a form suitable for administration to a patient. In the preferred embodiment, the pharmaceutical compositions are in a water soluble form, such as being present as pharmaceutically acceptable salts, which is meant to include both acid and base addition salts. "Pharmaceutically acceptable acid addition salt" refers to those salts that retain the biological effectiveness of the free bases and that are not biologically or otherwise undesirable, formed with inorganic acids such as hydrochloric acid, hydrobromic acid, sulfuric acid, nitric acid, phosphoric acid and the like, and organic acids such as acetic acid, propionic acid, glycolic acid, pyruvic acid, oxalic acid, maleic acid, malonic acid, succinic acid, fumaric acid, tartaric acid, citric acid, benzoic acid, cinnamic acid, mandelic acid, methanesulfonic acid, ethanesulfonic acid, p-toluenesulfonic acid, salicylic acid, and the like. "Pharmaceutically acceptable base addition salts" include those derived from inorganic bases such as sodium, potassium, lithium, ammonium, calcium, magnesium, iron, zinc, copper, manganese, aluminum salts and the like. Particularly preferred are the ammonium, potassium, sodium, calcium, and magnesium salts. Salts derived from pharmaceutically acceptable organic non-toxic bases include salts of primary, secondary, and tertiary amines, substituted amines including naturally occurring substituted amines, cyclic amines and basic ion exchange resins, such as isopropylamine, trimethylamine, diethylamine, triethylamine, tripropylamine, and ethanolamine.

The pharmaceutical compositions may also include one or more of the following: carrier proteins such as serum albumin; buffers; fillers such as microcrystalline cellulose, lactose, corn and other starches; binding agents; sweeteners and other flavoring agents; coloring agents; and polyethylene glycol.

The pharmaceutical compositions can be administered in a variety of unit dosage forms depending upon the method of administration. For example, unit dosage forms suitable for oral administration include, but are not limited to, powder, tablets, pills, capsules, and lozenges. It is recognized that ovarian cancer protein modulators (e.g., antibodies, antisense constructs, ribozymes, small organic molecules, etc.) when administered orally, should be protected from digestion. This is typically accomplished either by complexing the molecule(s) with a composition to render it resistant to acidic and enzymatic hydrolysis, or by packaging the molecule(s) in an appropriately resistant carrier, such as a liposome or a protection barrier. Means of protecting agents from digestion are well known in the art.

The compositions for administration will commonly comprise an ovarian cancer protein modulator dissolved in a pharmaceutically acceptable carrier, preferably an aqueous carrier. A variety of aqueous carriers can be used, e.g., buffered saline and the like. These solutions are sterile and generally free of undesirable matter. These compositions may be sterilized by conventional, well known sterilization techniques. The compositions may contain pharmaceutically acceptable auxiliary substances as required to approximate physiological conditions such as pH adjusting and buffering agents, toxicity adjusting agents and the like, e.g., sodium acetate, sodium chloride, potassium chloride, calcium chloride, sodium lactate and the like. The concentration of active agent in these formulations can vary widely, and will be selected primarily based on fluid volumes, viscosities, body weight, and the like in accordance with the particular mode of administration selected and the patient's needs. See, e.g., Remington's Pharmaceutical Science (15th ed., 1980) and Hardman and Limbird (eds. 2001) Goodman and Gillman: The Pharmacological Basis of Therapeutics (10th ed.) McGraw-Hill. Thus, a typical pharmaceutical composition for intravenous administration would be about 0.1 to 10 mg per patient per day. Dosages from 0.1 up to about 100 mg per patient per day may be used, particularly when the drug is administered to a secluded site and not into the blood stream, such as into a body cavity or into a lumen of an organ. Substantially higher dosages are possible in topical administration. Actual methods for preparing parenterally administrable compositions are readily available.

The compositions containing modulators of ovarian cancer proteins can be administered for therapeutic or prophylactic treatments. In therapeutic applications, compositions are administered to a patient suffering from a disease (e.g., a cancer) in an amount sufficient to cure or at least partially arrest the disease and/or its complications. An amount adequate to accomplish this is defined as a "therapeutically effective dose." Amounts effective for this use will depend upon the severity of the disease and the general state of the patient's health. Single or multiple administrations of the compositions may be administered depending on the dosage and frequency as required and tolerated by the patient. In any event, the composition should provide a sufficient quantity of the agents of this invention to effectively treat the patient. An amount of modulator that is capable of preventing or slowing the development of cancer in a mammal is referred to as a "prophylactically effective dose." The particular dose required for a prophylactic treatment will depend upon the medical condition and history of the mammal, the particular cancer being prevented, as well as other factors such as age, weight, gender, administration route, efficiency, etc. Such prophylactic

treatments may be used, e.g., in a mammal who has previously had cancer to prevent a recurrence of the cancer, or in a mammal who is suspected of having a significant likelihood of developing cancer based, e.g., in part, upon gene expression profiles. Vaccine strategies may be used, in either a DNA vaccine form, or protein vaccine.

It will be appreciated that the present ovarian cancer protein-modulating compounds can be administered alone or in combination with additional ovarian cancer modulating compounds or with other therapeutic agent, e.g., other anti-cancer agents or treatments.

In numerous embodiments, one or more nucleic acids, e.g., polynucleotides comprising nucleic acid sequences set forth in Tables 1-26, such as RNAi, antisense polynucleotides or ribozymes, will be introduced into cells, in vitro or in vivo. The present invention provides methods, reagents, vectors, and cells useful for expression of ovarian cancer-associated polypeptides and nucleic acids using in vitro (cell-free), ex vivo or in vivo (cell or organism-based) recombinant expression systems.

The particular procedure used to introduce the nucleic acids into a host cell for expression of a protein or nucleic acid is application specific. Many procedures for introducing foreign nucleotide sequences into host cells may be used. These include the use of calcium phosphate transfection, spheroplasts, electroporation, liposomes, microinjection, plasma vectors, viral vectors and any of the other well known methods for introducing cloned genomic DNA, cDNA, synthetic DNA or other foreign genetic material into a host cell. See, e.g., Berger and Kimmel (1987) Guide to Molecular Cloning Techniques from Methods in Enzymology (vol. 152) Academic Press; Ausubel, et al. (eds. 1999 and supplements) Current Protocols Lippincott; and Sambrook, et al. (2001) Molecular Cloning: A Laboratory Manual (3d ed., Vol. 1-3) CSH Press.

In a preferred embodiment, ovarian cancer proteins and modulators are administered as therapeutic agents, and can be formulated as outlined above. Similarly, ovarian cancer genes (including both the full-length sequence, partial sequences, or regulatory sequences of the ovarian cancer coding regions) can be administered in a gene therapy application. These ovarian cancer genes can include antisense applications, either as gene therapy (e.g., for incorporation into the genome) or as antisense compositions, as will be appreciated by those in the art.

Ovarian cancer polypeptides and polynucleotides can also be administered as vaccine compositions to stimulate HTL, CTL, and antibody responses. Such vaccine compositions can include, e.g., lipidated peptides (see, e.g., Vitiello, et al. (1995) J. Clin. Invest. 95:341-

349), peptide compositions encapsulated in poly(D,L-lactide-co-glycolide, "PLG") microspheres (see, e.g., Eldridge, et al. (1991) Molec. Immunol. 28:287-294; Alonso, et al. (1994) Vaccine 12:299-306; Jones, et al. (1995) Vaccine 13:675-681), peptide compositions contained in immune stimulating complexes (ISCOMS; see, e.g., Takahashi, et al. (1990) Nature 344:873-875; Hu, et al. (1998) Clin. Exp. Immunol. 113:235-243), multiple antigen peptide systems (MAPs; see, e.g., Tam (1988) Proc. Nat'l Acad. Sci. USA 85:5409-5413;

Tam (1996) J. Immunol. Methods 196:17-32), peptides formulated as multivalent peptides; peptides for use in ballistic delivery systems, typically crystallized peptides, viral delivery vectors (Perkus, et al., p. 379, in Kaufmann (ed. 1996) Concepts in Vaccine Development de

5 Gruyter; Chakrabarti, et al. (1986) Nature 320:535-537; Hu, et al. (1986) Nature 320:537-540; Kieny, et al. (1986) AIDS BioTechnology 4:790-795; Top, et al. (1971) J. Infect. Dis. 124:148-154; Chanda, et al. (1990) Virology 175:535-547), particles of viral or synthetic

10 origin (see, e.g., Kofler, et al. (1996) J. Immunol. Methods 192:25-35; Eldridge, et al. (1993) Sem. Hematol. 30:16-24; Falo, et al. (1995) Nature Med. 7:649-653), adjuvants (Warren, et al. (1986) Ann. Rev. Immunol. 4:369-388; Gupta, et al. (1993) Vaccine 11:293-306), liposomes (Reddy, et al. (1992) J. Immunol. 148:1585-1589; Rock (1996) Immunol. Today 17:131-137), or, naked or particle absorbed cDNA (Ulmer, et al. (1993) Science 259:1745-

15 1749; Robinson, et al. (1993) Vaccine 11:957-960; Shiver, et al., p. 423, in Kaufmann (ed. 1996) Concepts in Vaccine Development de Gruyter; Cease and Berzofsky (1994) Ann. Rev. Immunol. 12:923-989; and Eldridge, et al. (1993) Sem. Hematol. 30:16-24). Toxin-targeted

20 delivery technologies, also known as receptor mediated targeting, such as those of Avant Immunotherapeutics, Inc. (Needham, Massachusetts) may also be used.

Vaccine compositions often include adjuvants. Many adjuvants contain a substance designed to protect the antigen from rapid catabolism, such as aluminum hydroxide or

25 mineral oil, and a stimulator of immune responses, such as lipid A, Bordetella pertussis, or Mycobacterium tuberculosis derived proteins. Certain adjuvants are commercially available

as, e.g., Freund's Incomplete Adjuvant and Complete Adjuvant (Difco Laboratories, Detroit, MI); Merck Adjuvant 65 (Merck and Company, Inc., Rahway, NJ); AS-2 (SmithKline Beecham, Philadelphia, PA); aluminum salts such as aluminum hydroxide gel (alum) or

30 aluminum phosphate; salts of calcium, iron or zinc; an insoluble suspension of acylated tyrosine; acylated sugars; cationically or anionically derivatized polysaccharides;

polyphosphazenes; biodegradable microspheres; monophosphoryl lipid A and quil A.

Cytokines, such as GM-CSF, interleukin-2, -7, -12, and other like growth factors, may also be

used as adjuvants.

Vaccines can be administered as nucleic acid compositions wherein DNA or RNA encoding one or more of the polypeptides, or a fragment thereof, is administered to a patient. See, e.g., Wolff et al. (1990) *Science* 247:1465-1468; U.S. Patent Nos. 5,580,859; 5,589,466; 5,804,566; 5,739,118; 5,736,524; 5,679,647; and WO 98/04720. Examples of DNA-based delivery technologies include "naked DNA", facilitated (bupivacaine, polymers, peptide-mediated) delivery, cationic lipid complexes, and particle-mediated ("gene gun") or pressure-mediated delivery (see, e.g., U.S. Patent No. 5,922,687).

For therapeutic or prophylactic immunization purposes, the peptides of the invention can be expressed by viral or bacterial vectors. Examples of expression vectors include attenuated viral hosts, such as vaccinia or fowlpox. This approach involves the use of vaccinia virus, e.g., as a vector to express nucleotide sequences that encode ovarian cancer polypeptides or polypeptide fragments. Upon introduction into a host, the recombinant vaccinia virus expresses the immunogenic peptide, and thereby elicits an immune response. Vaccinia vectors and methods useful in immunization protocols are described in, e.g., U.S. Patent No. 4,722,848. Another vector is BCG (Bacille Calmette Guérin). BCG vectors are described in Stover, et al. (1991) *Nature* 351:456-460. A wide variety of other vectors useful for therapeutic administration or immunization e.g., adeno and adeno-associated virus vectors, retroviral vectors, *Salmonella typhi* vectors, detoxified anthrax toxin vectors, and the like, will be apparent. See, e.g., Shata, et al. (2000) *Mol. Med. Today* 6:66-71; Shedlock, et al. (2000) *J. Leukoc. Biol.* 68:793-806; and Hipp, et al. (2000) *In Vivo* 14:571-85.

Methods for the use of genes as DNA vaccines are well known, and include placing an ovarian cancer gene or portion of an ovarian cancer gene under the control of a regulatable promoter or a tissue-specific promoter for expression in an ovarian cancer patient. The ovarian cancer gene used for DNA vaccines can encode full-length ovarian cancer proteins, but more preferably encodes portions of the ovarian cancer proteins including peptides derived from the ovarian cancer protein. In one embodiment, a patient is immunized with a DNA vaccine comprising a plurality of nucleotide sequences derived from an ovarian cancer gene. For example, ovarian cancer-associated genes or sequence encoding subfragments of an ovarian cancer protein are introduced into expression vectors and tested for their immunogenicity in the context of Class I MHC and an ability to generate cytotoxic T cell responses. This procedure provides for production of cytotoxic T cell responses against cells which present antigen, including intracellular epitopes.

In a preferred embodiment, the DNA vaccines include a gene encoding an adjuvant molecule with the DNA vaccine. Such adjuvant molecules include cytokines that increase the immunogenic response to the ovarian cancer polypeptide encoded by the DNA vaccine. Additional or alternative adjuvants are available.

In another preferred embodiment ovarian cancer genes find use in generating animal models of ovarian cancer. When the ovarian cancer gene identified is repressed or diminished in cancer tissue, gene therapy technology, e.g., wherein antisense RNA directed to the ovarian cancer gene will also diminish or repress expression of the gene. Animal models of ovarian cancer find use in screening for modulators of an ovarian cancer-associated sequence or modulators of ovarian cancer. Similarly, transgenic animal technology including gene knockout technology, e.g., as a result of homologous recombination with an appropriate gene targeting vector, will result in the absence or increased expression of the ovarian cancer protein. When desired, tissue-specific expression or knockout of the ovarian cancer protein may be necessary.

It is also possible that the ovarian cancer protein is overexpressed in ovarian cancer. As such, transgenic animals can be generated that overexpress the ovarian cancer protein. Depending on the desired expression level, promoters of various strengths can be employed to express the transgene. Also, the number of copies of the integrated transgene can be determined and compared for a determination of the expression level of the transgene. Animals generated by such methods find use as animal models of ovarian cancer and are additionally useful in screening for modulators to treat ovarian cancer.

Kits for Use in Diagnostic and/or Prognostic Applications

For use in diagnostic, research, and therapeutic applications suggested above, kits are also provided by the invention. In the diagnostic and research applications such kits may include any or all of the following: assay reagents, buffers, ovarian cancer-specific nucleic acids or antibodies, hybridization probes and/or primers, siRNA or antisense polynucleotides, ribozymes, dominant negative ovarian cancer polypeptides or polynucleotides, small molecule inhibitors of ovarian cancer-associated sequences etc. A therapeutic product may include sterile saline or another pharmaceutically acceptable emulsion and suspension base.

In addition, the kits may include instructional materials containing directions (e.g., protocols) for the practice of the methods of this invention. While the instructional materials typically comprise written or printed materials they are not limited to such. Any medium

capable of storing such instructions and communicating them to an end user is contemplated by this invention. Such media include, but are not limited to electronic storage media (e.g., magnetic discs, tapes, cartridges, chips), optical media (e.g., CD ROM), and the like. Such media may include addresses to internet sites that provide such instructional materials.

- 5 The present invention also provides for kits for screening for modulators of ovarian cancer-associated sequences. Such kits can be prepared from readily available materials and reagents. For example, such kits can comprise one or more of the following materials: an ovarian cancer-associated polypeptide or polynucleotide, reaction tubes, and instructions for testing ovarian cancer-associated activity. Optionally, the kit contains biologically active ovarian cancer protein. A wide variety of kits and components can be prepared according to the present invention, depending upon the intended user of the kit and the particular needs of the user. Diagnosis would typically involve evaluation of a plurality of genes or products. The genes will be selected based on correlations with important parameters in disease which may be identified in historical or outcome data.

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EXAMPLES

Example 1: Gene Chip Analysis

- 20 Molecular profiles of various normal and cancerous tissues were determined and analyzed using gene chips. RNA was isolated and gene chip analysis was performed as described (Glynn, et al. (2000) *Nature* 403:672-676; Zhao, et al. (2000) *Genes Dev.* 14:981-993).

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TABLE 1A lists about 1119 genes up-regulated in ovarian cancer compared to normal adult tissues. These were selected from 5900 probes on the Affymetrix GeneChip array such that the ratio of "average" ovarian cancer to "average" normal adult tissues was greater than or equal to 5.0. This "average" ovarian cancer level was set to be the 80th percentile value amongst various ovarian cancers. The "average" normal adult tissue level was set to be the 80th percentile amongst various non-malignant tissues.

TABLE 1A: ABOUT 1119 UP-REGULATED OVARIAN CANCER GENES

Probe	Accession	Gene	Ratio
42384	AF055928	hsp70	63.7
42387	AF178161	hsp70	63.7
42388	AF178161	hsp70	63.7
42389	AF178161	hsp70	63.7
42390	AF178161	hsp70	63.7
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42666	AF178161	hsp70	

[illegible][illegible]

[illegible]

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29556	AW27455	Hs.29545	ES1a	"
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29649	A43841.38	Hs.29649	ES1a	"
29650	A43841.38	Hs.29650	ES1a	"
29651	A43841.38	Hs.29651	ES1a	"
29652	A43841.38	Hs.29652	ES1a	"
29653	A43841.38	Hs.29653	ES1a	"
29654	A43841.38	Hs.29654	ES1a	"
29655	A43841.38	Hs.29655	ES1a	"
29656	A43841.38	Hs.29656	ES1a	"
29657	A43841.38	Hs.29657	ES1a	"
29658	A43841.38	Hs.29658	ES1a	"
29659	A43841.38	Hs.29659	ES1a	"
29660	A43841.38	Hs.29660	ES1a	"
29661	A43841.38	Hs.29661	ES1a	"
29662	A43841.38	Hs.29662	ES1a	"
29663	A43841.38	Hs.29663	ES1a	"
29664	A43841.38	Hs.29664	ES1a	"
29665	A43841.38	Hs.29665	ES1a	"
29666	A43841.38	Hs.29666	ES1a	"
29667	A43841.38	Hs.29667	ES1a	"
29668	A43841.38	Hs.29668	ES1a	"
29669	A43841.38	Hs.29669	ES1a	"
29670	A43841.38	Hs.29670	ES1a	"
29671	A43841.38	Hs.29671	ES1a	"
29672	A43841.38	Hs.29672	ES1a	"
29673	A43841.38	Hs.29673	ES1a	"
29674	A43841.38	Hs.29674	ES1a	"
29675	A43841.38	Hs.29675	ES1a	"
29676	A43841.38	Hs.29676	ES1a	"
29677	A43841.38	Hs.29677	ES1a	"
29678	A43841.38	Hs.29678	ES1a	"
29679	A43841.38	Hs.29679	ES1a	"
29680	A43841.38	Hs.29680	ES1a	"
29681	A43841.38	Hs.29681	ES1a	"
29682	A43841.38	Hs.29682	ES1a	"
29683	A43841.38	Hs.29683	ES1a	"
29684	A43841.38	Hs.29684	ES1a	"
29685	A43841.38	Hs.29685	ES1a	"
29686	A43841.38	Hs.29686	ES1a	"
29687	A43841.38	Hs.29687	ES1a	"
29688	A43841.38	Hs.29688	ES1a	"
29689	A43841.38	Hs.29689	ES1a	"
29690	A43841.38	Hs.29690	ES1a	"
29691	A43841.38	Hs.29691	ES1a	"
29692	A43841.38	Hs.29692	ES1a	"
29693	A43841.38	Hs.29693	ES1a	"
29694	A43841.38	Hs.29694	ES1a	"
29695	A43841.38	Hs.29695	ES1a	"
29696	A43841.38	Hs.29696	ES1a	"
29697	A43841.38	Hs.29697	ES1a	"
29698	A43841.38	Hs.29698	ES1a	"
29699	A43841.38	Hs.29699	ES1a	"
29700	A43841.38	Hs.29700	ES1a	"

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[illegible]

[illegible]

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TABLE 2A lists about 187 genes up-regulated in ovarian cancer compared to normal adult tissues that are likely to be extracellular or cell-surface proteins. These were selected as per Table 1A, except that the ratio was greater than or equal to 2.5, and the predicted protein contained a PFAM domain that is a cell-surface protein (e.g., Ig, Ig-like, etc.).

TABLE 2B lists about 187 up-regulated ONCOGENE GENES ENCODING EXTRACELLULAR/CELL SURFACE PROTEINS

Ex. Acc. No. UG ID Title

1 403741 7330932 Mmus 7833-468

2 403742 7330933 Mmus 7833-469

3 403743 7330934 Mmus 7833-470

4 403744 7330935 Mmus 7833-471

5 403745 7330936 Mmus 7833-472

6 403746 7330937 Mmus 7833-473

7 403747 7330938 Mmus 7833-474

8 403748 7330939 Mmus 7833-475

9 403749 7330940 Mmus 7833-476

10 403750 7330941 Mmus 7833-477

11 403751 7330942 Mmus 7833-478

12 403752 7330943 Mmus 7833-479

13 403753 7330944 Mmus 7833-480

14 403754 7330945 Mmus 7833-481

15 403755 7330946 Mmus 7833-482

16 403756 7330947 Mmus 7833-483

17 403757 7330948 Mmus 7833-484

18 403758 7330949 Mmus 7833-485

19 403759 7330950 Mmus 7833-486

20 403760 7330951 Mmus 7833-487

21 403761 7330952 Mmus 7833-488

22 403762 7330953 Mmus 7833-489

23 403763 7330954 Mmus 7833-490

24 403764 7330955 Mmus 7833-491

25 403765 7330956 Mmus 7833-492

26 403766 7330957 Mmus 7833-493

27 403767 7330958 Mmus 7833-494

28 403768 7330959 Mmus 7833-495

29 403769 7330960 Mmus 7833-496

30 403770 7330961 Mmus 7833-497

31 403771 7330962 Mmus 7833-498

32 403772 7330963 Mmus 7833-499

33 403773 7330964 Mmus 7833-500

34 403774 7330965 Mmus 7833-501

35 403775 7330966 Mmus 7833-502

36 403776 7330967 Mmus 7833-503

37 403777 7330968 Mmus 7833-504

38 403778 7330969 Mmus 7833-505

39 403779 7330970 Mmus 7833-506

40 403780 7330971 Mmus 7833-507

41 403781 7330972 Mmus 7833-508

42 403782 7330973 Mmus 7833-509

43 403783 7330974 Mmus 7833-510

44 403784 7330975 Mmus 7833-511

45 403785 7330976 Mmus 7833-512

46 403786 7330977 Mmus 7833-513

47 403787 7330978 Mmus 7833-514

48 403788 7330979 Mmus 7833-515

49 403789 7330980 Mmus 7833-516

50 403790 7330981 Mmus 7833-517

51 403791 7330982 Mmus 7833-518

52 403792 7330983 Mmus 7833-519

53 403793 7330984 Mmus 7833-520

54 403794 7330985 Mmus 7833-521

55 403795 7330986 Mmus 7833-522

56 403796 7330987 Mmus 7833-523

57 403797 7330988 Mmus 7833-524

58 403798 7330989 Mmus 7833-525

59 403799 7330990 Mmus 7833-526

60 403800 7330991 Mmus 7833-527

61 403801 7330992 Mmus 7833-528

62 403802 7330993 Mmus 7833-529

63 403803 7330994 Mmus 7833-530

64 403804 7330995 Mmus 7833-531

65 403805 7330996 Mmus 7833-532

66 403806 7330997 Mmus 7833-533

67 403807 7330998 Mmus 7833-534

68 403808 7330999 Mmus 7833-535

69 403809 7331000 Mmus 7833-536

70 403810 7331001 Mmus 7833-537

71 403811 7331002 Mmus 7833-538

72 403812 7331003 Mmus 7833-539

73 403813 7331004 Mmus 7833-540

74 403814 7331005 Mmus 7833-541

75 403815 7331006 Mmus 7833-542

76 403816 7331007 Mmus 7833-543

77 403817 7331008 Mmus 7833-544

78 403818 7331009 Mmus 7833-545

79 403819 7331010 Mmus 7833-546

80 403820 7331011 Mmus 7833-547

81 403821 7331012 Mmus 7833-548

82 403822 7331013 Mmus 7833-549

83 403823 7331014 Mmus 7833-550

84 403824 7331015 Mmus 7833-551

85 403825 7331016 Mmus 7833-552

86 403826 7331017 Mmus 7833-553

87 403827 7331018 Mmus 7833-554

88 403828 7331019 Mmus 7833-555

89 403829 7331020 Mmus 7833-556

90 403830 7331021 Mmus 7833-557

91 403831 7331022 Mmus 7833-558

92 403832 7331023 Mmus 7833-559

93 403833 7331024 Mmus 7833-560

94 403834 7331025 Mmus 7833-561

95 403835 7331026 Mmus 7833-562

96 403836 7331027 Mmus 7833-563

97 403837 7331028 Mmus 7833-564

98 403838 7331029 Mmus 7833-565

99 403839 7331030 Mmus 7833-566

100 403840 7331031 Mmus 7833-567

101 403841 7331032 Mmus 7833-568

102 403842 7331033 Mmus 7833-569

103 403843 7331034 Mmus 7833-570

104 403844 7331035 Mmus 7833-571

105 403845 7331036 Mmus 7833-572

106 403846 7331037 Mmus 7833-573

107 403847 7331038 Mmus 7833-574

108 403848 7331039 Mmus 7833-575

109 403849 7331040 Mmus 7833-576

110 403850 7331041 Mmus 7833-577

111 403851 7331042 Mmus 7833-578

112 403852 7331043 Mmus 7833-579

113 403853 7331044 Mmus 7833-580

114 403854 7331045 Mmus 7833-581

115 403855 7331046 Mmus 7833-582

116 403856 7331047 Mmus 7833-583

117 403857 7331048 Mmus 7833-584

118 403858 7331049 Mmus 7833-585

[illegible]

[illegible]

Ha.21443	EST1	FLJ174055 (Hs_MGC_17) Homo sapiens cDNA clone IMAGE 485465
Ha.21443	EST1	FLJ174055 (Hs_MGC_17) Homo sapiens cDNA clone IMAGE 485465
Ha.02960	EST1	Homo sapiens cDNA FLJ11341 la, clone PLACE100
Ha.85026	EST1	hypothetical protein FLJ11382
Ha.85424	predicted exon	hypothetical (desminogen-binding protein)
Ha.28555	predicted exon	Homo sapiens cDNA FLJ21425 la, clone HFC08658
Ha.50684	EST1	carcinoembryonic antigen-related cell adhesion molecule 1 (CEACAM1)
Ha.233952	EST1	protein kinase (source, monophosphatase), alpha type, 7
Ha.76412	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.65403	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.169149	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.167904	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.42925	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.14848	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.102416	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.21093	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.1447	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.153275	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.237356	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.57573	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.159207	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.76065	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.207355	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.139204	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.139205	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.77573	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.18141	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.203674	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.6838	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.158146	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.46818	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.65403	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.131388	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.37903	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.33981	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.278692	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.79120	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.27553	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.65553	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.182485	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.33476	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.239177	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.9379	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.209061	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.31123	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.67776	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49505	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49506	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49507	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49508	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49509	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49510	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49511	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49512	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49513	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49514	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49515	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49516	EST1	phosphatase (source, monophosphatase), alpha type, 7
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Ha.49518	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49519	EST1	phosphatase (source, monophosphatase), alpha type, 7
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Ha.49522	EST1	phosphatase (source, monophosphatase), alpha type, 7
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Ha.49524	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49525	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49526	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49527	EST1	phosphatase (source, monophosphatase), alpha type, 7
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Ha.49530	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49531	EST1	phosphatase (source, monophosphatase), alpha type, 7
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Ha.49535	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49536	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49537	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49538	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49539	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49540	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49541	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49542	EST1	phosphatase (source, monophosphatase), alpha type, 7
Ha.49543	EST1	phosphatase (source

[illegible]

[illegible][illegible]

[illegible][illegible]

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41724	AA70569	Hs.71618	polymerase (RNA) I (DNA directed) polypeptide L7.	18.5
41943	AA05869	Hs.20186	EST, Weakly similar to Yhc21p [S. cerevisiae]	18.5
41967	AB38569	Hs.27569	EST	18.5
42356	AW02337	Hs.15849	EST	18.5
41712	X18696	Hs.81412	Proteinase M89	18.5
41825	BE299161	Hs.75564	Interleukin 1 receptor, type I	18.4
42367	AW06823	Hs.15206	Insulin-like growth factor binding protein 7	18.4
43351	A71235	Hs.1470	IL-1RI receptor	18.4
44069	B517892	Hs.695	actin related protein 20 complex, subunit 3 [2140]	18.4
44277	AA05879	Hs.18528	EST	18.4
44344	AA58133	Hs.20186	EST	18.4
44807	B52116	Hs.15384	binding motif protein 3	18.4
44873	AA34551	Hs.24662	fibronectin-related protein	18.4
44952			predicted exon	18.4
45059			predicted exon	18.4
45096	AU76803	Hs.25275	carboxylesterase 2 (intestinal, liver)	18.3
46044	NL.01238	Hs.25574	oligosaccharide receptor, family 2, subfamily C, member 1	18.3
46111	AA14119	Hs.82222	sera domain, immunoglobulin domain (Ig), short basic	18.3
46449			predicted exon	18.3
46490	AL134791	Hs.17440	mRNA for FLJ10023 protein	18.3
46542	BE35342	Hs.25492	EST, Weakly similar to CLUE, HUMAN CLAUDIN	18.3
46711	AW17377	Hs.17440	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.3
46728	AT13816	Hs.77348	hypoxanthine phosphoribosyl transferase (HGPRT)	18.3
46911	H5393	Hs.10571	cathepsin D (lysosomal aspartic protease)	18.2
46957	BE27342	Hs.24661	EST	18.2
46959			predicted exon	18.2
46960	NL.003378	Hs.20795	hemoglobin, beta pseudogene 1	18.2
46961	AA54688	Hs.171014	VGF nerve growth factor inducible	18.2
46962	AA54688	Hs.171014	beaded filament structural protein 2, plakophilin	18.2
46963	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46964	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46965	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46966	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46967	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46968	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46969	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46970	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46971	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46972	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46973	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46974	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46975	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46976	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46977	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46978	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46979	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46980	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46981	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46982	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46983	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46984	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46985	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46986	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46987	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46988	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46989	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46990	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46991	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46992	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46993	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46994	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46995	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46996	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46997	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46998	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
46999	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47000	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47001	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47002	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47003	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47004	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47005	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47006	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47007	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47008	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47009	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47010	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47011	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47012	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
47013	AA54688	Hs.171014	gpcr-4/CTD12c-19085-007-409 CT125 Homo sapiens	18.2
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Plus	714945	55498-59268
Minus	5923630	162607-82000
Plus	7342161	310-42
Minus	6602635	6905-7072
Plus	9586030	4785-5400
Minus	9712335	84306-84635
Plus	9256205	85153-85277
Minus	9375793	10966-14506
Plus	9785568	4056643-1092931
Minus	7711478	32504-32017
Plus	7711622	55454-65912,68118-66586
Plus	005160	
Minus	403027	
Plus	403215	
Minus	40328	
Plus	406277	
Minus	40638	
Plus	40638	
Minus	40638	
Plus	406457	
Minus	406473	
Plus	406531	
Minus	40657	

ABR1-E4A tests about 131 genes up-regulated in ovarian cancer compared to normal ovaries that are likely to be extracellular or cell-surface proteins. These were selected as for stable 3A, except that the ratio was greater than or equal to 10, and the predicted protein contained a P-AM domain that is indicative of extracellular localization.

[illegible]

TABLE 4B:
Key: Unique Eos probe set identifier number
CAT number: Gene cluster number
Accessions: GenBank accession numbers

Play	CAT Number	Accession
110498	120811.1	AA355749 AW085520 AW965633 AA340319 BE1705936
110441	159480.1	BE457197 AA1162474 AA180369 BE275628 BE776131
121258	200725.1	AA258731 AA257621 AW188228 AW137774
131705	279993.1	AW040750 AW040753 TR0540 AA111969

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PCT/US02/19397

42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																		
HA0257	HA0258	HA0259	HA0260	HA0261	HA0262	HA0263	HA0264	HA0265	HA0266	HA0267	HA0268	HA0269	HA0270	HA0271	HA0272	HA0273	HA0274	HA0275	HA0276	HA0277	HA0278	HA0279	HA0280	HA0281	HA0282	HA0283	HA0284	HA0285	HA0286	HA0287	HA0288	HA0289	HA0290	HA0291	HA0292	HA0293	HA0294	HA0295	HA0296	HA0297	HA0298	HA0299	HA0300	HA0301	HA0302	HA0303	HA0304	HA0305	HA0306	HA0307	HA0308	HA0309	HA0310	HA0311	HA0312	HA0313	HA0314	HA0315	HA0316	HA0317	HA0318	HA0319	HA0320	HA0321	HA0322	HA0323	HA0324	HA0325	HA0326	HA0327	HA0328	HA0329	HA0330	HA0331	HA0332	HA0333	HA0334	HA0335	HA0336	HA0337	HA0338	HA0339	HA0340	HA0341	HA0342	HA0343	HA0344	HA0345	HA0346	HA0347	HA0348	HA0349	HA0350	HA0351	HA0352	HA0353	HA0354	HA0355	HA0356	HA0357	HA0358	HA0359	HA0360	HA0361	HA0362	HA0363	HA0364	HA0365	HA0366	HA0367	HA0368	HA0369	HA0370	HA0371	HA0372	HA0373	HA0374	HA0375	HA0376	HA0377	HA0378	HA0379	HA0380	HA0381	HA0382	HA0383	HA0384	HA0385	HA0386	HA0387	HA0388	HA0389	HA0390	HA0391	HA0392	HA0393	HA0394	HA0395	HA0396	HA0397	HA0398	HA0399	HA0400	HA0401	HA0402	HA0403	HA0404	HA0405	HA0406	HA0407	HA0408	HA0409	HA0410	HA0411	HA0412	HA0413	HA0414	HA0415	HA0416	HA0417	HA0418	HA0419	HA0420	HA0421	HA0422	HA0423	HA0424	HA0425	HA0426	HA0427	HA0428	HA0429	HA0430	HA0431	HA0432	HA0433	HA0434	HA0435	HA0436	HA0437	HA0438	HA0439	HA0440	HA0441	HA0442	HA0443	HA0444	HA0445	HA0446	HA0447	HA0448	HA0449	HA0450	HA0451	HA0452	HA0453	HA0454	HA0455	HA0456	HA0457	HA0458	HA0459	HA0460	HA0461	HA0462	HA0463	HA0464	HA0465	HA0466	HA0467	HA0468	HA0469	HA0470	HA0471	HA0472	HA0473	HA0474	HA0475	HA0476	HA0477	HA0478	HA0479	HA0480	HA0481	HA0482	HA0483	HA0484	HA0485	HA0486	HA0487	HA0488	HA0489	HA0490	HA0491	HA0492	HA0493	HA0494	HA0495	HA0496	HA0497	HA0498	HA0499	HA0500	HA0501	HA0502	HA0503	HA0504	HA0505	HA0506	HA0507	HA0508	HA0509	HA0510	HA0511	HA0512	HA0513	HA0514	HA0515	HA0516	HA0517	HA0518	HA0519	HA0520	HA0521	HA0522	HA0523	HA0524	HA0525	HA0526	HA0527	HA0528	HA0529	HA0530	HA0531	HA0532	HA0533	HA0534	HA0535	HA0536	HA0537	HA0538	HA0539	HA0540	HA0541	HA0542	HA0543	HA0544	HA0545	HA0546	HA0547	HA0548	HA0549	HA0550	HA0551	HA0552	HA0553	HA0554	HA0555	HA0556	HA0557	HA0558	HA0559	HA0560	HA0561	HA0562	HA0563	HA0564	HA0565	HA0566	HA0567	HA0568	HA0569	HA0570	HA0571	HA0572	HA0573	HA0574	HA0575	HA0576	HA0577	HA0578	HA0579	HA0580	HA0581	HA0582	HA0583	HA0584	HA0585	HA0586	HA0587	HA0588	HA0589	HA0590	HA0591	HA0592	HA0593	HA0594	HA0595	HA0596	HA0597	HA0598	HA0599	HA0600	HA0601	HA0602	HA0603	HA0604	HA0605	HA0606	HA0607	HA0608	HA0609	HA0610	HA0611	HA0612	HA0613	HA0614	HA0615	HA0616	HA0617	HA0618	HA0619	HA0620	HA0621	HA0622	HA0623	HA0624	HA0625	HA0626	HA0627	HA0628	HA0629	HA0630	HA0631	HA0632	HA0633	HA0634	HA0635	HA0636	HA0637	HA0638	HA0639	HA0640	HA0641	HA0642	HA0643	HA0644	HA0645	HA0646	HA0647	HA0648	HA0649	HA0650	HA0651	HA0652	HA0653	HA0654	HA0655	HA0656	HA0657	HA0658	HA0659	HA0660	HA0661	HA0662	HA0663	HA0664	HA0665	HA0666	HA0667	HA0668	HA0669	HA0670	HA0671	HA0672	HA0673	HA0674	HA0675	HA0676	HA0677	HA0678	HA0679	HA0680	HA0681	HA0682	HA0683	HA0684	HA0685	HA0686	HA0687	HA0688	HA0689	HA0690	HA0691	HA0692	HA0693	HA0694	HA0695	HA0696	HA0697	HA0698	HA0699	HA0700	HA0701	HA0702	HA0703	HA0704	HA0705	HA0706	HA0707	HA0708	HA0709	HA0710	HA0711	HA0712	HA0713	HA0714	HA0715	HA0716	HA0717	HA0718	HA0719	HA0720	HA0721	HA0722	HA0723	HA0724	HA0725	HA0726	HA0727	HA0728	HA0729	HA0730	HA0731	HA0732	HA0733	HA0734	HA0735	HA0736	HA0737	HA0738	HA0739	HA0740	HA0741	HA0742	HA0743	HA0744	HA0745	HA0746	HA0747	HA0748	HA0749	HA0750	HA0751	HA0752	HA0753	HA0754	HA0755	HA0756	HA0757	HA0758	HA0759	HA0760	HA0761	HA0762	HA0763	HA0764	HA0765	HA0766	HA0767	HA0768	HA0769	HA0770	HA0771	HA0772	HA0773	HA0774	HA0775	HA0776	HA0777	HA0778	HA0779	HA0780	HA0781	HA0782	HA0783	HA0784	HA0785	HA0786	HA0787	HA0788	HA0789	HA0790	HA0791	HA0792	HA0793	HA0794	HA0795	HA0796	HA0797	HA0798	HA0799	HA0800	HA0801	HA0802	HA0803	HA0804	HA0805	HA0806	HA0807	HA0808	HA0809	HA0810	HA0811	HA0812	HA0813	HA0814	HA0815	HA0816	HA0817	HA0818	HA0819	HA0820	HA0821	HA0822	HA0823	HA0824	HA0825	HA0826	HA0827	HA0828	HA0829	HA0830	HA0831	HA0832	HA0833	HA0834	HA0835	HA0836	HA0837	HA0838	HA0839	HA0840	HA0841	HA0842	HA0843	HA0844	HA0845	HA0846	HA0847	HA0848	HA0849	HA0850	HA0851	HA0852	HA0853	HA0854	HA0855	HA0856	HA0857	HA0858	HA0859	HA0860	HA0861	HA0862	HA0863	HA0864	HA0865	HA0866	HA0867	HA0868	HA0869	HA0870	HA0871	HA0872	HA0873	HA0874	HA0875	HA0876	HA0877	HA0878	HA0879	HA0880	HA0881	HA0882	HA0883	HA0884	HA0885	HA0886	HA0887	HA0888	HA0889	HA0890	HA0891	HA0892	HA0893	HA0894	HA0895	HA0896	HA0897	HA0898	HA0899	HA0900	HA0901	HA0902	HA0903	HA0904	HA0905	HA0906	HA0907	HA0908	HA0909	HA0910	HA0911	HA0912	HA0913	HA0914	HA0915	HA0916	HA0917	HA0918	HA0919	HA0920	HA0921	HA0922	HA0923	HA0924	HA0925	HA0926	HA0927	HA0928	HA0929	HA0930	HA0931	HA0932	HA0933	HA0934	HA0935	HA0936	HA0937	HA0938	HA0939	HA0940	HA0941	HA0942	HA0943	HA0944	HA0945	HA0946	HA0947	HA0948	HA0949	HA0950	HA0951	HA0952	HA0953	HA0954	HA0955	HA0956	HA0957	HA0958	HA0959	HA0960	HA0961	HA0962	HA0963	HA0964	HA0965	HA0966	HA0967	HA0968	HA0969	HA0970	HA0971	HA0972	HA0973	HA0974	HA0975	HA0976	HA0977	HA0978	HA0979	HA0980	HA0981	HA0982	HA0983	HA0984	HA0985	HA0986	HA0987	HA0988	HA0989	HA0990	HA0991	HA0992	HA0993	HA0994	HA0995	HA0996	HA0997	HA0998	HA0999	HA1000	HA1001	HA1002	HA1003	HA1004	HA1005	HA1006	HA1007	HA1008	HA1009	HA1010	HA1011	HA1012	HA1013	HA1014	HA1015	HA1016	HA1017	HA1018	HA1019	HA1020	HA1021	HA1022	HA1023	HA1024	HA1025	HA1026	HA1027	HA1028	HA1029	HA1030	HA1031	HA1032	HA1033	HA1034	HA1035	HA1036	HA1037	HA1038	HA1039	HA1040	HA1041	HA1042	HA1043	HA1044	HA1045	HA1046	HA1047	HA1048	HA1049	HA1050	HA1051	HA1052	HA1053	HA1054	HA1055	HA1056	HA1057	HA1058	HA1059	HA1060	HA1061	HA1062	HA1063	HA1064	HA1065	HA1066	HA1067	HA1068	HA1069	HA1070	HA1071	HA1072	HA1073	HA1074	HA1075	HA1076	HA1077	HA1078	HA1079	HA1080	HA1081	HA1082	HA1083	HA1084	HA1085	HA1086	HA1087	HA1088	HA1089	HA1090	HA1091	HA1092	HA1093	HA1094	HA1095	HA1096	HA1097	HA1098	HA1099	HA1100	HA1101	HA1102	HA1103	HA1104	HA1105	HA1106	HA1107	HA1108	HA1109	HA1110	HA1111	HA1112	HA1113	HA1114	HA1115	HA1116	HA1117	HA1118	HA1119	HA1120	HA1121	HA1122	HA1123	HA1124	HA1125	HA1126	HA1127	HA1128	HA1129	HA1130	HA1131	HA1132	HA1133	HA1134	HA1135	HA1136	HA1137	HA1138	HA1139	HA1140	HA1141	HA1142	HA1143	HA1144	HA1145	HA1146	HA1147	HA1148	HA1149	HA1150	HA1151	HA1152	HA1153	HA1154	HA1155	HA1156	HA1157	HA1158	HA1159	HA1160	HA1161	HA1162	HA1163	HA1164	HA1165	HA1166	HA1167	HA1168	HA1169	HA1170	HA1171	HA1172	HA1173	HA1174	HA1175	HA1176	HA1177	HA1178	HA1179	HA1180	HA1181	HA1182	HA1183	HA1184	HA1185	HA1186	HA1187	HA1188	HA1189	HA1190	HA1191	HA1192	HA1193	HA1194	HA1195	HA1196	HA1197	HA1198	HA1199	HA1200	HA1201	HA1202	HA1203	HA1204	HA1205	HA1206	HA1207	HA1208	HA1209	HA1210	HA1211	HA1212	HA1213	HA1214	HA1215	HA1216	HA1217	HA1218	HA1219	HA1220	HA1221	HA1222	HA1223	HA1224	HA1225	HA1226	HA1227	HA1228	HA1229	HA1230	HA1231	HA1232	HA1233	HA1234	HA1235	HA1236	HA1237	HA1238	HA1239	HA1240	HA1241	HA1242	HA1243	HA1244	HA1245	HA1246	HA1247	HA1248	HA1249	HA1250	HA1251	HA1252	HA1253	HA1254	HA1255	HA1256	HA1257	HA1258	HA1259	HA1260	HA1261	HA1262	HA1263	HA1264	HA1265	HA1266	HA1267	HA1268	HA1269	HA1270	HA1271	HA1272	HA1273	HA1274	HA1275	HA1276	HA1277	HA1278	HA1279	HA1280	HA1281	HA1282	HA1283	HA1284	HA1285	HA1286	HA1287	HA1288	HA1289	HA1290	HA1291	HA1292	HA1293	HA1294	HA1295	HA1296	HA1297	HA1298	HA1299	HA1300	HA1301	HA1302	HA1303	HA1304	HA1305	HA1306	HA1307	HA1308	HA1309	HA1310	HA1311	HA1312	HA1313	HA1314	HA1315	HA1316	HA1317	HA1318	HA1319	HA1320	HA1321	HA1322	HA1323	HA1324	HA1325	HA1326	HA1327	HA1328	HA1329	HA1330	HA1331	HA1332	HA1333	HA1334	HA1335	HA1336	HA1337	HA1338	HA1339	HA1340	HA1341	HA1342	HA1343	HA1344	HA1345	HA1346	HA1347	HA1348	HA1349	HA1350	HA1351	HA1352	HA1353	HA1354	HA1355	HA1356	HA1357	HA1358	HA1359	HA1360	HA1361	HA1362	HA1363	HA1364	HA1365	HA1366	HA1367	HA1368	HA1369	HA1370	HA1371	HA1372	HA1373	HA1374	HA1375	HA1376	HA1377	HA1378	HA1379	HA1380	HA1381	HA1382	HA1383	HA1384	HA1385	HA1386	HA1387	HA1388	HA1389	HA1390	HA1391	HA1392	HA1393	HA1394	HA1395	HA1396	HA1397	HA1398	HA1399	HA1400	HA1401	HA1402	HA1403	HA1404	HA1405	HA1406	HA1407	HA1408	HA1409	HA1410	HA1411	HA1412	HA1413	HA1414	HA1415	HA1416	HA1417	HA1418	HA1419	HA1420	HA1421	HA1422	HA1423	HA1424	HA1425	HA1426	HA1427	HA1428	HA1429	HA1430	HA1431	HA1432	HA1433	HA1434	HA1435	HA1436	HA1437	HA1438	HA1439	HA1440	HA1441	HA1442	HA1443	HA1444	HA1445	HA1446	HA1447	HA1448	HA1449	HA1450	HA1451	HA1452	

41	IP10; Small inducible cytosolic subfamily B (
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AA065359	AA065360	AA065361	AA065362	AA065363	AA065364	AA065365	AA065366	AA065367	AA065368	AA065369	AA065370	AA065371	AA065372	AA065373	AA065374	AA065375	AA065376	AA065377	AA065378	AA065379	AA065380	AA065381	AA065382	AA065383	AA065384	AA065385	AA065386	AA065387	AA065388	AA065389	AA065390	AA065391	AA065392	AA065393	AA065394	AA065395	AA065396	AA065397	AA065398	AA065399	AA065400	AA065401	AA065402	AA065403	AA065404	AA065405	AA065406	AA065407	AA065408	AA065409	AA065410	AA065411	AA065412	AA065413	AA065414	AA065415	AA065416	AA065417	AA065418	AA065419	AA065420	AA065421	AA065422	AA065423	AA065424	AA065425	AA065426	AA065427	AA065428	AA065429	AA065430	AA065431	AA065432	AA065433	AA065434	AA065435	AA065436	AA065437	AA065438	AA065439	AA065440	AA065441	AA065442	AA065443	AA065444	AA065445	AA065446	AA065447	AA065448	AA065449	AA065450	AA065451	AA065452	AA065453	AA065454	AA065455	AA065456	AA065457	AA065458	AA065459	AA065460	AA065461	AA065462	AA065463	AA065464	AA065465	AA065466	AA065467	AA065468	AA065469	AA065470	AA065471	AA065472	AA065473	AA065474	AA065475	AA065476	AA065477	AA065478	AA065479	AA065480	AA065481	AA065482	AA065483	AA065484	AA065485	AA065486	AA065487	AA065488	AA065489	AA065490	AA065491	AA065492	AA065493	AA065494	AA065495	AA065496	AA065497	AA065498	AA065499	AA065500	AA065501	AA065502	AA065503	AA065504	AA065505	AA065506	AA065507	AA065508	AA065509	AA065510	AA065511	AA065512	AA065513	AA065514	AA065515	AA065516	AA065517	AA065518	AA065519	AA065520	AA065521	AA065522	AA065523	AA065524	AA065525	AA065526	AA065527	AA065528	AA065529	AA065530	AA065531	AA065532	AA065533	AA065534	AA065535	AA065536	AA065537	AA065538	AA065539	AA065540	AA065541	AA065542	AA065543	AA065544	AA065545	AA065546	AA065547	AA065548	AA065549	AA065550	AA065551	AA065552	AA065553	AA065554	AA065555	AA065556	AA065557	AA065558	AA065559	AA065560	AA065561	AA065562	AA065563	AA065564	AA065565	AA065566	AA065567	AA065568	AA065569	AA065570	AA065571	AA065572	AA065573	AA065574	AA065575	AA065576	AA065577	AA065578	AA065579	AA065580	AA065581	AA065582	AA065583	AA065584	AA065585	AA065586	AA065587	AA065588	AA065589	AA065590	AA065591	AA065592	AA065593	AA065594	AA065595	AA065596	AA065597	AA065598	AA065599	AA065600	AA065601	AA065602	AA065603	AA065604	AA065605	AA065606	AA065607	AA065608	AA065609	AA065610	AA065611	AA065612	AA065613	AA065614	AA065615	AA065616	AA065617	AA065618	AA065619	AA065620	AA065621	AA065622	AA065623	AA065624	AA065625	AA065626	AA065627	AA065628	AA065629	AA065630	AA065631	AA065632	AA065633	AA065634	AA065635	AA065636	AA065637	AA065638	AA065639	AA065640	AA065641	AA065642	AA065643	AA065644	AA065645	AA065646	AA065647	AA065648	AA065649	AA065650	AA065651	AA065652	AA065653	AA065654	AA065655	AA065656	AA065657	AA065658	AA065659	AA065660	AA065661	AA065662	AA065663	AA065664	AA065665	AA065666	AA065667	AA065668	AA065669	AA065670	AA065671	AA065672	AA065673	AA065674	AA065675	AA065676	AA065677	AA065678	AA065679	AA065680	AA065681	AA065682	AA065683	AA065684	AA065685	AA065686	AA065687	AA065688	AA065689	AA065690	AA065691	AA065692	AA065693	AA065694	AA065695	AA065696	AA065697	AA065698	AA065699	AA065700	AA065701	AA065702	AA065703	AA065704	AA065705	AA065706	AA065707	AA065708	AA065709	AA065710	AA065711	AA065712	AA065713	AA065714	AA065715	AA065716	AA065717	AA065718	AA065719	AA065720	AA065721	AA065722	AA065723	AA065724	AA065725	AA065726	AA065727	AA065728	AA065729	AA065730	AA065731	AA065732	AA065733	AA065734	AA065735	AA065736	AA065737	AA065738	AA065739	AA065740	AA065741	AA065742	AA065743	AA065744	AA065745	AA065746	AA065747	AA065748	AA065749	AA065750	AA065751	AA065752	AA065753	AA065754	AA065755	AA065756	AA065757	AA065758	AA065759	AA065760	AA065761	AA065762	AA065763	AA065764	AA065765	AA065766	AA065767	AA065768	AA065769	AA065770	AA065771	AA065772	AA065773	AA065774	AA065775	AA065776	AA065777	AA065778	AA065779	AA065780	AA065781	AA065782	AA065783	AA065784	AA065785	AA065786	AA065787	AA065788	AA065789	AA065790	AA065791	AA065792	AA065793	AA065794	AA065795	AA065796	AA065797	AA065798	AA065799	AA065800	AA065801	AA065802	AA065803	AA065804	AA065805	AA065806	AA065807	AA065808	AA065809	AA065810	AA065811	AA065812	AA065813	AA065814	AA065815	AA065816	AA065817	AA065818	AA065819	AA065820	AA065821	AA065822	AA065823	AA065824	AA065825	AA065826	AA065827	AA065828	AA065829	AA065830	AA065831	AA065832	AA065833	AA065834	AA065835	AA065836	AA065837	AA065838	AA065839	AA065840	AA065841	AA065842	AA065843	AA065844	AA065845	AA065846	AA065847	AA065848	AA065849	AA065850	AA065851	AA065852	AA065853	AA065854	AA065855	AA065856	AA065857	AA065858	AA065859	AA065860	AA065861	AA065862	AA065863	AA065864	AA065865	AA065866	AA065867	AA065868	AA065869	AA065870	AA065871	AA065872	AA065873	AA065874	AA065875	AA065876	AA065877	AA065878	AA065879	AA065880	AA065881	AA065882	AA065883	AA065884	AA065885	AA065886	AA065887	AA065888	AA065889	AA065890	AA065891	AA065892	AA065893	AA065894	AA065895	AA065896	AA065897	AA065898	AA065899	AA065900	AA065901	AA065902	AA065903	AA065904	AA065905	AA065906	AA065907	AA065908	AA065909	AA065910	AA065911	AA065912	AA065913	AA065914	AA065915	AA065916	AA065917	AA065918	AA065919	AA065920	AA065921	AA065922	AA065923	AA065924	AA065925	AA065926	AA065927	AA065928	AA065929	AA065930	AA065931	AA065932	AA065933	AA065934	AA065935	AA065936	AA065937	AA065938	AA065939	AA065940	AA065941	AA065942	AA065943	AA065944	AA065945	AA065946	AA065947	AA065948	AA065949	AA065950	AA065951	AA065952	AA065953	AA065954	AA065955	AA065956	AA065957	AA065958	AA065959	AA065960	AA065961	AA065962	AA065963	AA065964	AA065965	AA065966	AA065967	AA065968	AA065969	AA065970	AA065971	AA065972	AA065973	AA065974	AA065975	AA065976	AA065977	AA065978	AA065979	AA065980	AA065981	AA065982	AA065983	AA065984	AA065985	AA065986	AA065987	AA065988	AA065989	AA065990	AA065991	AA065992	AA065993	AA065994	AA065995	AA065996	AA065997	AA065998	AA065999	AA066000	AA066001	AA066002	AA066003	AA066004	AA066005	AA066006	AA066007	AA066008	AA066009	AA066010	AA066011	AA066012	AA066013	AA066014	AA066015	AA066016	AA066017	AA066018	AA066019	AA066020	AA066021	AA066022	AA066023	AA066024	AA066025	AA066026	AA066027	AA066028	AA066029	AA066030	AA066031	AA066032	AA066033	AA066034	AA066035	AA066036	AA066037	AA066038	AA066039	AA066040	AA066041	AA066042	AA066043	AA066044	AA066045	AA066046	AA066047	AA066048	AA066049	AA066050	AA066051	AA066052	AA066053	AA066054	AA066055	AA066056	AA066057	AA066058	AA066059	AA066060	AA066061	AA066062	AA066063	AA066064	AA066065	AA066066	AA066067	AA066068	AA066069	AA066070	AA066071	AA066072	AA066073	AA066074	AA066075	AA066076	AA066077	AA066078	AA066079	AA066080	AA066081	AA066082	AA066083	AA066084	AA066085	AA066086	AA066087	AA066088	AA066089	AA066090	AA066091	AA066092	AA066093	AA066094	AA066095	AA066096	AA066097	AA066098	AA066099	AA066100	AA066101	AA066102	AA066103	AA066104	AA066105	AA066106	AA066107	AA066108	AA066109	AA066110	AA066111	AA066112	AA066113	AA066114	AA066115	AA066116	AA066117	AA066118	AA066119	AA066120	AA066121	AA066122	AA066123	AA066124	AA066125	AA066126	AA066127	AA066128	AA066129	AA066130	AA066131	AA066132	AA066133	AA066134	AA066135	AA066136	AA066137	AA066138	AA066139	AA066140	AA066141	AA066142	AA066143	AA066144	AA066145	AA066146	AA066147	AA066148	AA066149	AA066150	AA066151	AA066152	AA066153	AA066154	AA066155	AA066156	AA066157	AA066158	AA066159	AA066160	AA066161	AA066162	AA066163	AA066164	AA066165	AA066166	AA066167	AA066168	AA066169	AA066170	AA066171	AA066172	AA066173	AA066174	AA066175	AA066176	AA066177	AA066178	AA066179	AA066180	AA066181	AA066182	AA066183	AA066184	AA066185	AA066186	AA066187	AA066188	AA066189	AA066190	AA066191	AA066192	AA066193	AA066194	AA066195	AA066196	AA066197	AA066198	AA066199	AA066200	AA066201	AA066202	AA066203	AA066204	AA066205	AA066206	AA066207	AA066208	AA066209	AA066210	AA066211	AA066212	AA066213	AA066214	AA066215	AA066216	AA066217	AA066218	AA066219	AA066220	AA066221	AA066222	AA066223	AA066224	AA066225	AA066226	AA066227	AA066228	AA066229	AA066230	AA066231	AA066232	AA066233	AA066234	AA066235	AA066236	AA066237	AA066238	AA066239	AA066240	AA066241	AA066242	AA066243	AA066244	AA066245	AA066246	AA066247	AA066248	AA066249	AA066250	AA066251	AA066252	AA066253	AA066254	AA066255	AA066256	AA066257	AA066258	AA066259	AA066260	AA066261	AA066262	AA066263	AA066264	AA066265	AA066266	AA066267	AA066268	AA066269	AA066270	AA066271	AA066272	AA066273	AA066274	AA066275	AA066276	AA066277	AA066278	AA066279	AA066280	AA066281	AA066282	AA066283	AA066284	AA066285	AA066286	AA066287	AA066288	AA066289	AA066290	AA066291	AA066292	AA066293	AA066294	AA066295	AA066296	AA066297	AA066298	AA066299	AA066300	AA066301	AA066302	AA066303	AA066304	AA066305	AA066306	AA066307	AA066308	AA066309	AA066310	AA066311	AA066312	AA066313	AA066314	AA066315	AA066316	AA066317	AA066318	AA066319	AA066320	AA066321	AA066322	AA066323	AA066324	AA066325	AA066326	AA066327	AA066328	AA066329	AA066330	AA066331	AA066332	AA066333	AA066334	AA066335	AA066336	AA066337	AA066338	AA066339	AA066340	AA066341	AA066342	AA066343	AA066344	AA066345	AA066346	AA066347	AA066348	AA066349	AA066350	AA066351	AA066352	AA066353	AA066354	AA066355	AA066356	AA066357	AA066358	AA066359	AA066360	AA066361	AA066362	AA066363	AA066364	AA066365	AA066366	AA066367	AA066368	AA066369	AA066370	AA066371	AA066372	AA066373	AA066374	AA066375	AA066376	AA066377	AA066378	AA066379	AA066380	AA066381	AA066382	AA066383	AA066384	AA066385	AA066386	AA066387	AA066388	AA066389	AA06639
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[illegible]

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[illegible][illegible]

[illegible]

[illegible]

Taken by this about 322 genes up-regulated in ovarian cancer compared to normal ovaries. These were selected from 35433 probesets on the Affymetrix U95-Hu1 GeneChip array such that the ratio of "average" ovarian cancer to "average" normal ovary values was greater than or equal to 10. The "average" ovarian cancer level was set to be the 2nd highest amongst the values for ovarian cancer. The "average" normal ovary level was set to be the 10th lowest amongst the values for non-malignant ovaries. In order to remove gene-specific background levels of non-specific hybridization, the 15th percentile value amongst the non-malignant tissues (see Table 7A) was subtracted from both the numerator and the denominator before the ratio was calculated.

TABLE 9A: 382 UP-REGULATED GENES, OVARIAN CANCER VERSUS NORMAL OVARY

[illegible]

5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80
101185	L19872	101192	101193	101194	101195	101196	101197	101198	101199	101200	101201	101202	101203	101204	101205
101206	101207	101208	101209	101210	101211	101212	101213	101214	101215	101216	101217	101218	101219	101220	101221
101222	101223	101224	101225	101226	101227	101228	101229	101230	101231	101232	101233	101234	101235	101236	101237
101238	101239	101240	101241	101242	101243	101244	101245	101246	101247	101248	101249	101250	101251	101252	101253
101254	101255	101256	101257	101258	101259	101260	101261	101262	101263	101264	101265	101266	101267	101268	101269
101270	101271	101272	101273	101274	101275	101276	101277	101278	101279	101280	101281	101282	101283	101284	101285
101286	101287	101288	101289	101290	101291	101292	101293	101294	101295	101296	101297	101298	101299	101300	101301
101302	101303	101304	101305	101306	101307	101308	101309	101310	101311	101312	101313	101314	101315	101316	101317
101318	101319	101320	101321	101322	101323	101324	101325	101326	101327	101328	101329	101330	101331	101332	101333
101334	101335	101336	101337	101338	101339	101340	101341	101342	101343	101344	101345	101346	101347	101348	101349
101350	101351	101352	101353	101354	101355	101356	101357	101358	101359	101360	101361	101362	101363	101364	101365
101366	101367	101368	101369	101370	101371	101372	101373	101374	101375	101376	101377	101378	101379	101380	101381
101382	101383	101384	101385	101386	101387	101388	101389	101390	101391	101392	101393	101394	101395	101396	101397
101398	101399	101400	101401	101402	101403	101404	101405	101406	101407	101408	101409	101410	101411	101412	101413
101414	101415	101416	101417	101418	101419	101420	101421	101422	101423	101424	101425	101426	101427	101428	101429
101430	101431	101432	101433	101434	101435	101436	101437	101438	101439	101440	101441	101442	101443	101444	101445
101446	101447	101448	101449	101450	101451	101452	101453	101454	101455	101456	101457	101458	101459	101460	101461
101462	101463	101464	101465	101466	101467	101468	101469	101470	101471	101472	101473	101474	101475	101476	101477
101478	101479	101480	101481	101482	101483	101484	101485	101486	101487	101488	101489	101490	101491	101492	101493
101494	101495	101496	101497	101498	101499	101500	101501	101502	101503	101504	101505	101506	101507	101508	101509
101510	101511	101512	101513	101514	101515	101516	101517	101518	101519	101520	101521	101522	101523	101524	101525
101526	101527	101528	101529	101530	101531	101532	101533	101534	101535	101536	101537	101538	101539	101540	101541
101542	101543	101544	101545	101546	101547	101548	101549	101550	101551	101552	101553	101554	101555	101556	101557
101558	101559	101560	101561	101562	101563	101564	101565	101566	101567	101568	101569	101570	101571	101572	101573
101574	101575	101576	101577	101578	101579	101580	101581	101582	101583	101584	101585	101586	101587	101588	101589
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172227	A0181268	Hs_123249	Human rat fibronectin type-3 and -5	
172228	T03351	Hs_123251	hA000141 Action Fast Cellulose fibers sapling	
172229	T03352	Hs_123252	Human cDNA sequence from clone 3504 on chromosome	
5	151982	A0433948	Hs_105203	ESTs. Moderately similar to KIAA0454 protein
123114	A0488407	Hs_105203	ESTs	
123442	A0588800	Hs_105203	ESTs	
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131941	D02637	Hs_35582	chitinolytic-specific protease 1	
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172231	A0181268	Hs_123249	Human rat fibronectin type-3 and -5	
172232	T03351	Hs_123251	hA000141 Action Fast Cellulose fibers sapling	
172233	T03352	Hs_123252	Human cDNA sequence from clone 3504 on chromosome	
5	151982	A0433948	Hs_105203	ESTs. Moderately similar to KIAA0454 protein
123114	A0488407	Hs_105203	ESTs	
123442	A0588800	Hs_105203	ESTs	
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172235	A0181268	Hs_123249	Human rat fibronectin type-3 and -5	
172236	T03351	Hs_123251	hA000141 Action Fast Cellulose fibers sapling	
172237	T03352	Hs_123252	Human cDNA sequence from clone 3504 on chromosome	
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123114	A0488407	Hs_105203	ESTs	
123442	A0588800	Hs_105203	ESTs	
123333	A0504253	Hs_105151	ESTs	
123689	A0069556	Hs_35582	ESTs	
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131942	D02638	Hs_35587	ESTs	
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172239	A0181268	Hs_123249	Human rat fibronectin type-3 and -5	
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172241	T03352	Hs_123252	Human cDNA sequence from clone 3504 on chromosome	
5	151982	A0433948	Hs_105203	ESTs. Moderately similar to KIAA0454 protein
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131942	D02638	Hs_35587	ESTs	
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Phylo: Unique Eco probe set identifier number
CAT: Gene cluster number
Accession: Genbank accession numbers

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	42135	BE320464	Hs.80316	ESTs	9.1
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	42140	BE320464	Hs.80316	ESTs	9.1
	42141	BE320464	Hs.80316	ESTs	9.1
	42142	BE320464	Hs.80316	ESTs	9.1
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	42212	BE320464	Hs.80316	ESTs	9.1
	42213	BE320464	Hs.80316	ESTs	9.1
	42214	BE320464	Hs.80316	ESTs	9.1
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	42216	BE320464	Hs.80316	ESTs	9.1
	42217	BE320464	Hs.80316	ESTs	9.1
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	42275	BE320464	Hs.80316	ESTs	9.1
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	42282	BE320464	Hs.80316	ESTs	9.1
	42283	BE320464	Hs.80316	ESTs	9.1
	42284	BE320464	Hs.8031		

[illegible]

[illegible]

[illegible]

40	415330	U82601	Hs_75381	hullrich 8 (neovirus, zymo)	85.74,lypsh	3.3
	41197			predicted error	anEts	3.3
	43525	A4714428	Hs_25145	homo sapiens cDNA FLJ14127 fs.	TM	3.2
	452013	AF274140	Hs_13982	homo sapiens cDNA FLJ127 fs.	TM	3.2

[illegible]

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[illegible]

20	BE440142	Hs.2610	ligand receptor protein 194D	28.4	43042	A1595841	Hs.8254	hypothetical protein PR00059	22.1
21	Hs.6540	EST1	cellular protein	29.2	43043	AW159585	Hs.10878	hypothetical protein FLJ21820	22.2
22	AW159582	Hs.151144	EST1	29.2	43044	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.3
23	AW159582	Hs.151144	EST1	29.1	43045	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.4
24	AW159582	Hs.151144	EST1	29.1	43046	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.5
25	AW159582	Hs.151144	EST1	29.1	43047	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.6
26	AW159582	Hs.151144	EST1	29.1	43048	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.7
27	AW159582	Hs.151144	EST1	29.1	43049	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.8
28	AW159582	Hs.151144	EST1	29.1	43050	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.9
29	AW159582	Hs.151144	EST1	29.1	43051	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.0
30	AW159582	Hs.151144	EST1	29.1	43052	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.1
31	AW159582	Hs.151144	EST1	29.1	43053	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.2
32	AW159582	Hs.151144	EST1	29.1	43054	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.3
33	AW159582	Hs.151144	EST1	29.1	43055	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.4
34	AW159582	Hs.151144	EST1	29.1	43056	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.5
35	AW159582	Hs.151144	EST1	29.1	43057	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.6
36	AW159582	Hs.151144	EST1	29.1	43058	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.7
37	AW159582	Hs.151144	EST1	29.1	43059	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.8
38	AW159582	Hs.151144	EST1	29.1	43060	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.9
39	AW159582	Hs.151144	EST1	29.1	43061	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.0
40	AW159582	Hs.151144	EST1	29.1	43062	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.1
41	AW159582	Hs.151144	EST1	29.1	43063	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.2
42	AW159582	Hs.151144	EST1	29.1	43064	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.3
43	AW159582	Hs.151144	EST1	29.1	43065	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.4
44	AW159582	Hs.151144	EST1	29.1	43066	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.5
45	AW159582	Hs.151144	EST1	29.1	43067	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.6
46	AW159582	Hs.151144	EST1	29.1	43068	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.7
47	AW159582	Hs.151144	EST1	29.1	43069	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.8
48	AW159582	Hs.151144	EST1	29.1	43070	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.9
49	AW159582	Hs.151144	EST1	29.1	43071	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.0
50	AW159582	Hs.151144	EST1	29.1	43072	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.1
51	AW159582	Hs.151144	EST1	29.1	43073	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.2
52	AW159582	Hs.151144	EST1	29.1	43074	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.3
53	AW159582	Hs.151144	EST1	29.1	43075	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.4
54	AW159582	Hs.151144	EST1	29.1	43076	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.5
55	AW159582	Hs.151144	EST1	29.1	43077	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.6
56	AW159582	Hs.151144	EST1	29.1	43078	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.7
57	AW159582	Hs.151144	EST1	29.1	43079	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.8
58	AW159582	Hs.151144	EST1	29.1	43080	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	25.9
59	AW159582	Hs.151144	EST1	29.1	43081	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.0
60	AW159582	Hs.151144	EST1	29.1	43082	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.1
61	AW159582	Hs.151144	EST1	29.1	43083	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.2
62	AW159582	Hs.151144	EST1	29.1	43084	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.3
63	AW159582	Hs.151144	EST1	29.1	43085	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.4
64	AW159582	Hs.151144	EST1	29.1	43086	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.5
65	AW159582	Hs.151144	EST1	29.1	43087	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.6
66	AW159582	Hs.151144	EST1	29.1	43088	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.7
67	AW159582	Hs.151144	EST1	29.1	43089	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.8
68	AW159582	Hs.151144	EST1	29.1	43090	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	26.9
69	AW159582	Hs.151144	EST1	29.1	43091	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.0
70	AW159582	Hs.151144	EST1	29.1	43092	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.1
71	AW159582	Hs.151144	EST1	29.1	43093	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.2
72	AW159582	Hs.151144	EST1	29.1	43094	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.3
73	AW159582	Hs.151144	EST1	29.1	43095	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.4
74	AW159582	Hs.151144	EST1	29.1	43096	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.5
75	AW159582	Hs.151144	EST1	29.1	43097	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.6
76	AW159582	Hs.151144	EST1	29.1	43098	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.7
77	AW159582	Hs.151144	EST1	29.1	43099	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.8
78	AW159582	Hs.151144	EST1	29.1	43100	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	27.9
79	AW159582	Hs.151144	EST1	29.1	43101	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.0
80	AW159582	Hs.151144	EST1	29.1	43102	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.1
81	AW159582	Hs.151144	EST1	29.1	43103	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.2
82	AW159582	Hs.151144	EST1	29.1	43104	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.3
83	AW159582	Hs.151144	EST1	29.1	43105	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.4
84	AW159582	Hs.151144	EST1	29.1	43106	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.5
85	AW159582	Hs.151144	EST1	29.1	43107	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.6
86	AW159582	Hs.151144	EST1	29.1	43108	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.7
87	AW159582	Hs.151144	EST1	29.1	43109	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.8
88	AW159582	Hs.151144	EST1	29.1	43110	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	28.9
89	AW159582	Hs.151144	EST1	29.1	43111	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.0
90	AW159582	Hs.151144	EST1	29.1	43112	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.1
91	AW159582	Hs.151144	EST1	29.1	43113	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.2
92	AW159582	Hs.151144	EST1	29.1	43114	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.3
93	AW159582	Hs.151144	EST1	29.1	43115	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.4
94	AW159582	Hs.151144	EST1	29.1	43116	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.5
95	AW159582	Hs.151144	EST1	29.1	43117	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.6
96	AW159582	Hs.151144	EST1	29.1	43118	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.7
97	AW159582	Hs.151144	EST1	29.1	43119	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.8
98	AW159582	Hs.151144	EST1	29.1	43120	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	29.9
99	AW159582	Hs.151144	EST1	29.1	43121	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	30.0
100	AW159582	Hs.151144	EST1	29.1	43122	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	30.1

20	BE440142	Hs.2610	ligand receptor protein 194D	28.4	43042	A1595841	Hs.8254	hypothetical protein PR00059	22.1
21	Hs.6540	EST1	cellular protein	29.2	43043	AW159585	Hs.10878	hypothetical protein FLJ21820	22.2
22	AW159582	Hs.151144	EST1	29.2	43044	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.3
23	AW159582	Hs.151144	EST1	29.1	43045	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.4
24	AW159582	Hs.151144	EST1	29.1	43046	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.5
25	AW159582	Hs.151144	EST1	29.1	43047	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.6
26	AW159582	Hs.151144	EST1	29.1	43048	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.7
27	AW159582	Hs.151144	EST1	29.1	43049	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.8
28	AW159582	Hs.151144	EST1	29.1	43050	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	22.9
29	AW159582	Hs.151144	EST1	29.1	43051	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.0
30	AW159582	Hs.151144	EST1	29.1	43052	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.1
31	AW159582	Hs.151144	EST1	29.1	43053	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.2
32	AW159582	Hs.151144	EST1	29.1	43054	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.3
33	AW159582	Hs.151144	EST1	29.1	43055	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.4
34	AW159582	Hs.151144	EST1	29.1	43056	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.5
35	AW159582	Hs.151144	EST1	29.1	43057	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.6
36	AW159582	Hs.151144	EST1	29.1	43058	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.7
37	AW159582	Hs.151144	EST1	29.1	43059	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.8
38	AW159582	Hs.151144	EST1	29.1	43060	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	23.9
39	AW159582	Hs.151144	EST1	29.1	43061	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.0
40	AW159582	Hs.151144	EST1	29.1	43062	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.1
41	AW159582	Hs.151144	EST1	29.1	43063	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.2
42	AW159582	Hs.151144	EST1	29.1	43064	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.3
43	AW159582	Hs.151144	EST1	29.1	43065	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.4
44	AW159582	Hs.151144	EST1	29.1	43066	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.5
45	AW159582	Hs.151144	EST1	29.1	43067	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.6
46	AW159582	Hs.151144	EST1	29.1	43068	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.7
47	AW159582	Hs.151144	EST1	29.1	43069	AW159585	Hs.10458	small intestine cytosolic subunit A (Oxy-Oxy)	24.8
48	AW159582	Hs.151144	EST1	29.1	43070	AW159585	H		

5	43485	433771	Ha.82294	ESTs	18.1
	43486	433772	Ha.82295	ESTs	18.1
	43487	433773	Ha.82296	ESTs	18.1
	43488	433774	Ha.82297	ESTs	18.1
	43489	433775	Ha.82298	ESTs	18.1
	43490	433776	Ha.82299	ESTs	18.1
	43491	433777	Ha.82300	ESTs	18.1
	43492	433778	Ha.82301	ESTs	18.1
	43493	433779	Ha.82302	ESTs	18.1
	43494	433780	Ha.82303	ESTs	18.1
	43495	433781	Ha.82304	ESTs	18.1
	43496	433782	Ha.82305	ESTs	18.1
	43497	433783	Ha.82306	ESTs	18.1
	43498	433784	Ha.82307	ESTs	18.1
	43499	433785	Ha.82308	ESTs	18.1
	43500	433786	Ha.82309	ESTs	18.1
	43501	433787	Ha.82310	ESTs	18.1
	43502	433788	Ha.82311	ESTs	18.1
	43503	433789	Ha.82312	ESTs	18.1
	43504	433790	Ha.82313	ESTs	18.1
	43505	433791	Ha.82314	ESTs	18.1
	43506	433792	Ha.82315	ESTs	18.1
	43507	433793	Ha.82316	ESTs	18.1
	43508	433794	Ha.82317	ESTs	18.1
	43509	433795	Ha.82318	ESTs	18.1
	43510	433796	Ha.82319	ESTs	18.1
	43511	433797	Ha.82320	ESTs	18.1
	43512	433798	Ha.82321	ESTs	18.1
	43513	433799	Ha.82322	ESTs	18.1
	43514	433800	Ha.82323	ESTs	18.1
	43515	433801	Ha.82324	ESTs	18.1
	43516	433802	Ha.82325	ESTs	18.1
	43517	433803	Ha.82326	ESTs	18.1
	43518	433804	Ha.82327	ESTs	18.1
	43519	433805	Ha.82328	ESTs	18.1
	43520	433806	Ha.82329	ESTs	18.1
	43521	433807	Ha.82330	ESTs	18.1
	43522	433808	Ha.82331	ESTs	18.1
	43523	433809	Ha.82332	ESTs	18.1
	43524	433810	Ha.82333	ESTs	18.1
	43525	433811	Ha.82334	ESTs	18.1
	43526	433812	Ha.82335	ESTs	18.1
	43527	433813	Ha.82336	ESTs	18.1
	43528	433814	Ha.82337	ESTs	18.1
	43529	433815	Ha.82338	ESTs	18.1
	43530	433816	Ha.82339	ESTs	18.1
	43531	433817	Ha.82340	ESTs	18.1
	43532	433818	Ha.82341	ESTs	18.1
	43533	433819	Ha.82342	ESTs	18.1
	43534	433820	Ha.82343	ESTs	18.1
	43535	433821	Ha.82344	ESTs	18.1
	43536	433822	Ha.82345	ESTs	18.1
	43537	433823	Ha.82346	ESTs	18.1
	43538	433824	Ha.82347	ESTs	18.1
	43539	433825	Ha.82348	ESTs	18.1
	43540	433826	Ha.82349	ESTs	18.1
	43541	433827	Ha.82350	ESTs	18.1
	43542	433828	Ha.82351	ESTs	18.1
	43543	433829	Ha.82352	ESTs	18.1
	43544	433830	Ha.82353	ESTs	18.1
	43545	433831	Ha.82354	ESTs	18.1
	43546	433832	Ha.82355	ESTs	18.1

43735	A056929	Ha.15073	EST1	14.8	43901	W7365	Ha.124169	EST1
42435	A001390	Ha.15470	Homo sapiens cDNA FLJ10518 fs, clone NT67220	14.8	43902	B23532	Ha.251071	ATP synthase
42375	Ha.16343	EST1	EST1	14.8	43903	A03659	Ha.152071	ATP synthase
43801	Ha.28584	EST1	EST1	14.8	43904	Ha.152071	EST1	EST1
42485	Ha.28584	EST1	EST1	14.8	43905	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43906	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43907	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43908	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43909	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43910	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43911	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43912	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43913	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43914	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43915	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43916	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43917	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43918	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43919	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43920	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43921	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43922	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43923	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43924	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43925	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43926	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43927	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43928	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43929	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43930	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43931	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43932	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43933	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43934	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43935	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43936	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43937	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43938	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43939	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43940	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43941	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43942	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43943	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43944	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43945	Ha.152071	EST1	EST1
42511	Ha.28584	EST1	EST1	14.8	43946	Ha.152071	EST1	EST1

4107	AA07552	Ha.5086	ESTs	12.9	4010	AS95083	Ha.19329	0	ESTs	12.0
4108	AA07553	Ha.5087	ESTs	12.9	4011	AW01455	Ha.29564	0	ESTs	12.0
4207	AA07554	Ha.5088	ESTs	12.9	4012	BE19001	Ha.25300	0	ESTs	12.0
4208	AA07555	Ha.5089	ESTs	12.9	4013	BE19002	Ha.25301	0	ESTs	12.0
4209	AA07556	Ha.5090	ESTs	12.9	4014	BE19003	Ha.25302	0	ESTs	12.0
4210	AA07557	Ha.5091	ESTs	12.9	4015	BE19004	Ha.25303	0	ESTs	12.0
4211	AA07558	Ha.5092	ESTs	12.9	4016	BE19005	Ha.25304	0	ESTs	12.0
4212	AA07559	Ha.5093	ESTs	12.9	4017	BE19006	Ha.25305	0	ESTs	12.0
4213	AA07560	Ha.5094	ESTs	12.9	4018	BE19007	Ha.25306	0	ESTs	12.0
4214	AA07561	Ha.5095	ESTs	12.9	4019	BE19008	Ha.25307	0	ESTs	12.0
4215	AA07562	Ha.5096	ESTs	12.9	4020	BE19009	Ha.25308	0	ESTs	12.0
4216	AA07563	Ha.5097	ESTs	12.9	4021	BE19010	Ha.25309	0	ESTs	12.0
4217	AA07564	Ha.5098	ESTs	12.9	4022	BE19011	Ha.25310	0	ESTs	12.0
4218	AA07565	Ha.5099	ESTs	12.9	4023	BE19012	Ha.25311	0	ESTs	12.0
4219	AA07566	Ha.5100	ESTs	12.9	4024	BE19013	Ha.25312	0	ESTs	12.0
4220	AA07567	Ha.5101	ESTs	12.9	4025	BE19014	Ha.25313	0	ESTs	12.0
4221	AA07568	Ha.5102	ESTs	12.9	4026	BE19015	Ha.25314	0	ESTs	12.0
4222	AA07569	Ha.5103	ESTs	12.9	4027	BE19016	Ha.25315	0	ESTs	12.0
4223	AA07570	Ha.5104	ESTs	12.9	4028	BE19017	Ha.25316	0	ESTs	12.0
4224	AA07571	Ha.5105	ESTs	12.9	4029	BE19018	Ha.25317	0	ESTs	12.0
4225	AA07572	Ha.5106	ESTs	12.9	4030	BE19019	Ha.25318	0	ESTs	12.0
4226	AA07573	Ha.5107	ESTs	12.9	4031	BE19020	Ha.25319	0	ESTs	12.0
4227	AA07574	Ha.5108	ESTs	12.9	4032	BE19021	Ha.25320	0	ESTs	12.0
4228	AA07575	Ha.5109	ESTs	12.9	4033	BE19022	Ha.25321	0	ESTs	12.0
4229	AA07576	Ha.5110	ESTs	12.9	4034	BE19023	Ha.25322	0	ESTs	12.0
4230	AA07577	Ha.5111	ESTs	12.9	4035	BE19024	Ha.25323	0	ESTs	12.0
4231	AA07578	Ha.5112	ESTs	12.9	4036	BE19025	Ha.25324	0	ESTs	12.0
4232	AA07579	Ha.5113	ESTs	12.9	4037	BE19026	Ha.25325	0	ESTs	12.0
4233	AA07580	Ha.5114	ESTs	12.9	4038	BE19027	Ha.25326	0	ESTs	12.0
4234	AA07581	Ha.5115	ESTs	12.9	4039	BE19028	Ha.25327	0	ESTs	12.0
4235	AA07582	Ha.5116	ESTs	12.9	4040	BE19029	Ha.25328	0	ESTs	12.0
4236	AA07583	Ha.5117	ESTs	12.9	4041	BE19030	Ha.25329	0	ESTs	12.0
4237	AA07584	Ha.5118	ESTs	12.9	4042	BE19031	Ha.25330	0	ESTs	12.0
4238	AA07585	Ha.5119	ESTs	12.9	4043	BE19032	Ha.25331	0	ESTs	12.0
4239	AA07586	Ha.5120	ESTs	12.9	4044	BE19033	Ha.25332	0	ESTs	12.0
4240	AA07587	Ha.5121	ESTs	12.9	4045	BE19034	Ha.25333	0	ESTs	12.0
4241	AA07588	Ha.5122	ESTs	12.9	4046	BE19035	Ha.25334	0	ESTs	12.0
4242	AA07589	Ha.5123	ESTs	12.9	4047	BE19036	Ha.25335	0	ESTs	12.0
4243	AA07590	Ha.5124	ESTs	12.9	4048	BE19037	Ha.25336	0	ESTs	12.0
4244	AA07591	Ha.5125	ESTs	12.9	4049	BE19038	Ha.25337	0	ESTs	12.0
4245	AA07592	Ha.5126	ESTs	12.9	4050	BE19039	Ha.25338	0	ESTs	12.0
4246	AA07593	Ha.5127	ESTs	12.9	4051	BE19040	Ha.25339	0	ESTs	12.0
4247	AA07594	Ha.5128	ESTs	12.9	4052	BE19041	Ha.25340	0	ESTs	12.0
4248	AA07595	Ha.5129	ESTs	12.9	4053	BE19042	Ha.25341	0	ESTs	12.0
4249	AA07596	Ha.5130	ESTs	12.9	4054	BE19043	Ha.25342	0	ESTs	12.0
4250	AA07597	Ha.5131	ESTs	12.9	4055	BE19044	Ha.25343	0	ESTs	12.0
4251	AA07598	Ha.5132	ESTs	12.9	4056	BE19045	Ha.25344	0	ESTs	12.0
4252	AA07599	Ha.5133	ESTs	12.9	4057	BE19046	Ha.25345	0	ESTs	12.0
4253	AA07600	Ha.5134	ESTs	12.9	4058	BE19047	Ha.25346	0	ESTs	12.0
4254	AA07601	Ha.5135	ESTs	12.9	4059	BE19048	Ha.25347	0	ESTs	12.0
4255	AA07602	Ha.5136	ESTs	12.9	4060	BE19049	Ha.25348	0	ESTs	12.0
4256	AA07603	Ha.5137	ESTs	12.9	4061	BE19050	Ha.25349	0	ESTs	12.0
4257	AA07604	Ha.5138	ESTs	12.9	4062	BE19051	Ha.25350	0	ESTs	12.0
4258	AA07605	Ha.5139	ESTs	12.9	4063	BE19052	Ha.25351	0	ESTs	12.0
4259	AA07606	Ha.5140	ESTs	12.9	4064	BE19053	Ha.25352	0	ESTs	12.0
4260	AA07607	Ha.5141	ESTs	12.9	4065	BE19054	Ha.25353	0	ESTs	12.0
4261	AA07608	Ha.5142	ESTs	12.9	4066	BE19055	Ha.25354	0	ESTs	12.0
4262	AA07609	Ha.5143	ESTs	12.9	4067	BE19056	Ha.25355	0	ESTs	12.0
4263	AA07610	Ha.5144	ESTs	12.9	4068	BE19057	Ha.25356	0	ESTs	12.0
4264	AA07611	Ha.5145	ESTs	12.9	4069	BE19058	Ha.25357	0	ESTs	12.0
4265	AA07612	Ha.5146	ESTs	12.9	4070	BE19059	Ha.25358	0	ESTs	12.0
4266	AA07613	Ha.5147	ESTs	12.9	4071	BE19060	Ha.25359	0	ESTs	12.0
4267	AA07614	Ha.5148	ESTs	12.9	4072	BE19061	Ha.25360	0	ESTs	12.0
4268	AA07615	Ha.5149	ESTs	12.9	4073	BE19062	Ha.25361	0	ESTs	12.0
4269	AA07616	Ha.5150	ESTs	12.9	4074	BE19063	Ha.25362	0	ESTs	12.0
4270	AA07617	Ha.5151	ESTs	12.9	4075	BE19064	Ha.25363	0	ESTs	12.0
4271	AA07618	Ha.5152	ESTs	12.9	4076	BE19065	Ha.25364	0	ESTs	12.0
4272	AA07619	Ha.5153	ESTs	12.9	4077	BE19066	Ha.25365	0	ESTs	12.0
4273	AA07620	Ha.5154	ESTs	12.9	4078	BE19067	Ha.25366	0	ESTs	12.0
4274	AA07621	Ha.5155	ESTs	12.9	4079	BE19068	Ha.25367	0	ESTs	12.0
4275	AA07622	Ha.5156	ESTs	12.9	4080	BE19069	Ha.25368	0	ESTs	12.0
4276	AA07623	Ha.5157	ESTs	12.9	4081	BE19070	Ha.25369	0	ESTs	12.0
4277	AA07624	Ha.5158	ESTs	12.9	4082	BE19071	Ha.25370	0	ESTs	12.0
4278	AA07625	Ha.5159	ESTs	12.9	4083	BE19072	Ha.25371	0	ESTs	12.0
4279	AA07626	Ha.5160	ESTs	12.9	4084	BE19073	Ha.25372	0	ESTs	12.0
4280	AA07627	Ha.5161	ESTs	12.9	4085	BE19074	Ha.25373	0	ESTs	12.0
4281	AA07628	Ha.5162	ESTs	12.9	4086	BE19075	Ha.25374	0	ESTs	12.0
4282	AA07629	Ha.5163	ESTs	12.9	4087	BE19076	Ha.25375	0	ESTs	12.0
4283	AA07630	Ha.5164	ESTs	12.9	4088	BE19077	Ha.25376	0	ESTs	12.0
4284	AA07631	Ha.5165	ESTs	12.9	4089	BE19078	Ha.25377	0	ESTs	12.0
4285	AA07632	Ha.5166	ESTs	12.9	4090	BE19079	Ha.25378	0	ESTs	12.0
4286	AA07633	Ha.5167	ESTs	12.9	4091	BE19080	Ha.25379	0	ESTs	12.0
4287	AA07634	Ha.5168	ESTs	12.9	4092	BE19081	Ha.25380	0	ESTs	12.0
4288	AA07635	Ha.5169	ESTs	12.9	4093	BE19082	Ha.25381	0	ESTs	12.0
4289	AA07636	Ha.5170	ESTs	12.9	4094	BE19083	Ha.25382	0	ESTs	12.0
4290	AA07637	Ha.5171	ESTs	12.9	4095	BE19084	Ha.25383	0	ESTs	12.0
4291	AA07638	Ha.5172	ESTs	12.9	4096	BE19085	Ha.25384	0	ESTs	12.0
4292	AA07639	Ha.5173	ESTs	12.9	4097	BE19086	Ha.25385	0	ESTs	12.0
4293	AA07640	Ha.5174	ESTs	12.9	4098	BE19087	Ha.25386	0	ESTs	12.0
4294	AA07641	Ha.5175	ESTs	12.9	4099	BE19088	Ha.25387	0	ESTs	12.0
4295	AA07642	Ha.5176	ESTs	12.9	4100	BE19089	Ha.25388	0	ESTs	12.0
4296	AA07643	Ha.5177	ESTs	12.9	4101	BE19090	Ha.25389	0	ESTs	12.0
4297	AA07644	Ha.5178	ESTs	12.9	4102	BE19091	Ha.25390	0	ESTs	12.0
4298	AA07645	Ha.5179	ESTs	12.9	4103	BE19092	Ha.25391	0	ESTs	12.0
4299	AA07646	Ha.5180	ESTs	12.9	4104	BE19093	Ha.25392	0	ESTs	12.0
4300	AA07647	Ha.5181	ESTs	12.9	4105	BE19094	Ha.25393	0	ESTs	12.0
4301	AA07648	Ha.5182	ESTs	12.9	4106	BE19095	Ha.25394	0	ESTs	12.0
4302	AA07649	Ha.5183	ESTs	12.9	4107	BE19096	Ha.25395	0	ESTs	12.0
4303	AA07650	Ha.5184	ESTs	12.9	4108	BE19097	Ha.25396	0	ESTs	12.0
4304	AA07651	Ha.5185	ESTs	12.9	4109	BE19098	Ha.25397	0	ESTs	12.0
4305	AA07652	Ha.5186	ESTs	12.9	4110	BE19099	Ha.25398	0	ESTs	12.0
4306	AA07653	Ha.5187	ESTs	12.9	4111	BE19100	Ha.25399	0	ESTs	12.0
4307	AA07654	Ha.5188	ESTs	12.9	4112	BE19101	Ha.25400	0	ESTs	12.0
4308	AA07655	Ha.5189	ESTs	12.9	4113	BE19102	Ha.25401	0	ESTs	12.0
4309	AA07656	Ha.5190	ESTs	12.9	4114	BE19103	Ha.25402	0	ESTs	12.0
4310	AA07657	Ha.5191	ESTs	12.9	4115	BE19104	Ha.25403	0	ESTs	12.0
4311	AA07658	Ha.5192	ESTs	12.9	4116	BE19105	Ha.25404	0	ESTs	12.0
4312	AA07659	Ha.5193	ESTs	12.9	4117	BE19106	Ha.25405	0	ESTs	12.0
4313	AA07660	Ha.5194	ESTs	12.9	4118	BE19107	Ha.25406	0	ESTs	12.0
4314	AA07661	Ha.5195	ESTs	12.9	4119	BE19108	Ha.25407	0	ESTs	12.0
4315	AA07662	Ha.5196	ESTs	12.9	4120	BE19109	Ha.25408	0	ESTs	12.0
4316	AA07663	Ha.5197	ESTs	12.9	4121	BE19110	Ha.25409	0	ESTs	12.0
4317	AA07664	Ha.5198	ESTs	12.9	4122	BE19111	Ha.25410	0	ESTs	12.0
4318	AA07665	Ha.5199	ESTs	12.9	4123	BE19112	Ha			

[illegible]

432904	AW070602	Ha.105421	ESTa, Weekly similar to AF161840 1 CG-422 pro	0	ESTa, Weekly similar to AF161840 1 CG-422 pro	10.2
432905	R32032	Ha.176817	ESTa	0	ESTa	10.2
432906	BE338115	Ha.169693	ESTa	0	ESTa	10.2
432907	AB033101	Ha.102796	KIAA1278 protein	0	KIAA1278 protein	10.2
432908	BE333667	Ha.295953	ESTa, Weekly similar to AF220049 1 uncharacter	0	ESTa, Weekly similar to AF220049 1 uncharacter	10.2
432909	AW172209	Ha.249999	ESTa	0	ESTa	10.1
432910	BE337258	Ha.210336	ESTa	0	ESTa	10.1
432911	HA.22587	Ha.176817	ESTa	0	ESTa	10.1
432912	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432913	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432914	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432915	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432916	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432917	R32048	Ha.239868	ESTa	0	ESTa	10.1
432918	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432919	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432920	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432921	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432922	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432923	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432924	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432925	R32048	Ha.239868	ESTa	0	ESTa	10.1
432926	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432927	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432928	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432929	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432930	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432931	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432932	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432933	R32048	Ha.239868	ESTa	0	ESTa	10.1
432934	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432935	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432936	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432937	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432938	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432939	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432940	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432941	R32048	Ha.239868	ESTa	0	ESTa	10.1
432942	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432943	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432944	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432945	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432946	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432947	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432948	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432949	R32048	Ha.239868	ESTa	0	ESTa	10.1
432950	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432951	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432952	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432953	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432954	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432955	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432956	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432957	R32048	Ha.239868	ESTa	0	ESTa	10.1
432958	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432959	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432960	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432961	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432962	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432963	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432964	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432965	R32048	Ha.239868	ESTa	0	ESTa	10.1
432966	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432967	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432968	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432969	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432970	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432971	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432972	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432973	R32048	Ha.239868	ESTa	0	ESTa	10.1
432974	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432975	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432976	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432977	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432978	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432979	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432980	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432981	R32048	Ha.239868	ESTa	0	ESTa	10.1
432982	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432983	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432984	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432985	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432986	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432987	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432988	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432989	R32048	Ha.239868	ESTa	0	ESTa	10.1
432990	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432991	BE337258	Ha.176817	ESTa	0	ESTa	10.1
432992	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
432993	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
432994	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
432995	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
432996	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
432997	R32048	Ha.239868	ESTa	0	ESTa	10.1
432998	AW172209	Ha.210336	ESTa	0	ESTa	10.1
432999	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433000	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433001	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433002	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433003	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433004	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433005	R32048	Ha.239868	ESTa	0	ESTa	10.1
433006	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433007	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433008	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433009	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433010	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433011	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433012	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433013	R32048	Ha.239868	ESTa	0	ESTa	10.1
433014	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433015	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433016	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433017	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433018	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433019	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433020	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433021	R32048	Ha.239868	ESTa	0	ESTa	10.1
433022	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433023	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433024	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433025	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433026	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433027	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433028	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433029	R32048	Ha.239868	ESTa	0	ESTa	10.1
433030	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433031	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433032	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433033	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433034	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433035	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433036	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433037	R32048	Ha.239868	ESTa	0	ESTa	10.1
433038	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433039	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433040	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433041	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433042	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433043	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433044	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433045	R32048	Ha.239868	ESTa	0	ESTa	10.1
433046	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433047	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433048	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433049	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433050	AF151074	Ha.132744	hypothetical protein	0	hypothetical protein	10.1
433051	AW075168	Ha.13337	ESTa, Weekly similar to unannoted protein produ	0	ESTa, Weekly similar to unannoted protein produ	10.1
433052	HA.239818	Ha.239818	phosphatidyle-3-kinase, catalytic, beta po	0	phosphatidyle-3-kinase, catalytic, beta po	10.1
433053	R32048	Ha.239868	ESTa	0	ESTa	10.1
433054	AW172209	Ha.210336	ESTa	0	ESTa	10.1
433055	BE337258	Ha.176817	ESTa	0	ESTa	10.1
433056	AW044139	Ha.174424	ESTa, Weekly similar to p110delta [Muncastin]	0	ESTa, Weekly similar to p110delta [Muncastin]	10.1
433057	BE316787	Ha.16393	B-cell CLL/lymphoma 7B	0	B-cell CLL/lymphoma 7B	10.1
433058	AF151074	Ha.132744				

45493	154559.1	AMT15297	AMT15299	AMT15304	AMT15304	AMT15309
45493	154560.1	BE15301	BE15302	BE15303	BE15304	BE15305
45493	154561.1	BE15306	BE15307	BE15308	BE15309	BE15310
45493	154562.1	BE15311	BE15312	BE15313	BE15314	BE15315
45493	154563.1	BE15316	BE15317	BE15318	BE15319	BE15320
45493	154564.1	BE15321	BE15322	BE15323	BE15324	BE15325
45493	154565.1	BE15326	BE15327	BE15328	BE15329	BE15330
45493	154566.1	BE15331	BE15332	BE15333	BE15334	BE15335
45493	154567.1	BE15336	BE15337	BE15338	BE15339	BE15340
45493	154568.1	BE15341	BE15342	BE15343	BE15344	BE15345
45493	154569.1	BE15346	BE15347	BE15348	BE15349	BE15350
45493	154570.1	BE15351	BE15352	BE15353	BE15354	BE15355
45493	154571.1	BE15356	BE15357	BE15358	BE15359	BE15360
45493	154572.1	BE15361	BE15362	BE15363	BE15364	BE15365
45493	154573.1	BE15366	BE15367	BE15368	BE15369	BE15370
45493	154574.1	BE15371	BE15372	BE15373	BE15374	BE15375
45493	154575.1	BE15376	BE15377	BE15378	BE15379	BE15380
45493	154576.1	BE15381	BE15382	BE15383	BE15384	BE15385
45493	154577.1	BE15386	BE15387	BE15388	BE15389	BE15390
45493	154578.1	BE15391	BE15392	BE15393	BE15394	BE15395
45493	154579.1	BE15396	BE15397	BE15398	BE15399	BE15400
45493	154580.1	BE15401	BE15402	BE15403	BE15404	BE15405
45493	154581.1	BE15406	BE15407	BE15408	BE15409	BE15410
45493	154582.1	BE15411	BE15412	BE15413	BE15414	BE15415
45493	154583.1	BE15416	BE15417	BE15418	BE15419	BE15420
45493	154584.1	BE15421	BE15422	BE15423	BE15424	BE15425
45493	154585.1	BE15426	BE15427	BE15428	BE15429	BE15430
45493	154586.1	BE15431	BE15432	BE15433	BE15434	BE15435
45493	154587.1	BE15436	BE15437	BE15438	BE15439	BE15440
45493	154588.1	BE15441	BE15442	BE15443	BE15444	BE15445
45493	154589.1	BE15446	BE15447	BE15448	BE15449	BE15450
45493	154590.1	BE15451	BE15452	BE15453	BE15454	BE15455
45493	154591.1	BE15456	BE15457	BE15458	BE15459	BE15460
45493	154592.1	BE15461	BE15462	BE15463	BE15464	BE15465
45493	154593.1	BE15466	BE15467	BE15468	BE15469	BE15470
45493	154594.1	BE15471	BE15472	BE15473	BE15474	BE15475
45493	154595.1	BE15476	BE15477	BE15478	BE15479	BE15480
45493	154596.1	BE15481	BE15482	BE15483	BE15484	BE15485
45493	154597.1	BE15486	BE15487	BE15488	BE15489	BE15490
45493	154598.1	BE15491	BE15492	BE15493	BE15494	BE15495
45493	154599.1	BE15496	BE15497	BE15498	BE15499	BE15500
45493	154600.1	BE15501	BE15502	BE15503	BE15504	BE15505
45493	154601.1	BE15506	BE15507	BE15508	BE15509	BE15510
45493	154602.1	BE15511	BE15512	BE15513	BE15514	BE15515
45493	154603.1	BE15516	BE15517	BE15518	BE15519	BE15520
45493	154604.1	BE15521	BE15522	BE15523	BE15524	BE15525
45493	154605.1	BE15526	BE15527	BE15528	BE15529	BE15530
45493	154606.1	BE15531	BE15532	BE15533	BE15534	BE15535
45493	154607.1	BE15536	BE15537	BE15538	BE15539	BE15540
45493	154608.1	BE15541	BE15542	BE15543	BE15544	BE15545
45493	154609.1	BE15546	BE15547	BE15548	BE15549	BE15550
45493	154610.1	BE15551	BE15552	BE15553	BE15554	BE15555
45493	154611.1	BE15556	BE15557	BE15558	BE15559	BE15560
45493	154612.1	BE15561	BE15562	BE15563	BE15564	BE15565
45493	154613.1	BE15566	BE15567	BE15568	BE15569	BE15570
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45493	154619.1	BE15596	BE15597	BE15598	BE15599	BE15600
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[illegible]

EST1	Ha.1520412	77	very low density Receptor-like receptor	443838	Alu.02055	EST1	Ha.143779	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.173779	78	PTF-400 protein kinase 1	443839	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.57658	79	EST1	443840	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.53011	80	EST1	443841	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.48480	81	EST1	443842	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.127812	82	EST1	443843	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.24598	83	EST1	443844	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.197447	84	EST1	443845	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.153044	85	EST1	443846	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.43075	86	EST1	443847	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.44609	87	EST1	443848	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.24973	88	EST1	443849	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.46793	89	EST1	443850	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.14972	90	EST1	443851	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.58086	91	EST1	443852	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.1904	92	EST1	443853	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.15322	93	EST1	443854	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.403070	94	EST1	443855	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.100772	95	EST1	443856	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.134509	96	EST1	443857	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.42650	97	EST1	443858	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.65861	98	EST1	443859	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.14248	99	EST1	443860	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.102776	100	EST1	443861	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.82713	101	EST1	443862	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.133526	102	EST1	443863	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.10387	103	EST1	443864	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.29725	104	EST1	443865	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.55145	105	EST1	443866	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.29070	106	EST1	443867	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.89063	107	EST1	443868	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.15373	109	EST1	443870	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.355923	111	EST1	443872	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.240770	112	EST1	443873	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.97268	113	EST1	443874	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.11817	114	EST1	443875	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.89063	118	EST1	443879	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.257846	119	EST1	443880	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151700	120	EST1	443881	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.184768	122	EST1	443883	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.33426	124	EST1	443885	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.31322	125	EST1	443886	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.222194	127	EST1	443888	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.151460	168	EST1	443929	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
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EST1	Ha.151460	182	EST1	443943	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	183	EST1	443944	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	184	EST1	443945	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	185	EST1	443946	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	186	EST1	443947	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	187	EST1	443948	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H500612
EST1	Ha.151460	188	EST1	443949	Alu.02055	EST1	Ha.153012	Homo sapiens cDNA FLJ223411 fs, clone H5006

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411514	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411515	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411516	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411517	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411518	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411519	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411520	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411521	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411522	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411523	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411524	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411525	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411526	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411527	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411528	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411529	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411530	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411531	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411532	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411533	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411534	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411535	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411536	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411537	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411538	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411539	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411540	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411541	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411542	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411543	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411544	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411545	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411546	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411547	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411548	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411549	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411550	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411551	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411552	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411553	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411554	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411555	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411556	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411557	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411558	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411559	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411560	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411561	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411562	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411563	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411564	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411565	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411566	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411567	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411568	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411569	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411570	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411571	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411572	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411573	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411574	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411575	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411576	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411577	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411578	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411579	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411580	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411581	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411582	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411583	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411584	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411585	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411586	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411587	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411588	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411589	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411590	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411591	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411592	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411593	WZ2723	EST1	Ha.20155	EST1	Ha.20155	EMG1	5.1
411594	WZ2723	EST1	Ha.20155	EST1			

[illegible]

TABLE 148:
Pkey: Unique EcoS probeSet Identifier number
CAT number: Gene cluster number
Accession: Genbank accession numbers

Play	CAT Number	Accession
409773	110951.1	AA053019 AA14482
410174	121005.1	AW002001 BE070750 BE62940
411580	129443.1	AW051186 AW955967 BE114358
414245	158985.1	F025981 AA129371 AA133740 AW19878
414315	143512.1	TA16781 AA420658 F13554 AA49240 AA143177
414378	174556.1	AW042001 AA218051 AA234237
416804	178138.1	AA083552 AB974745 AA136350 AA077465
418393	183793.1	AA683593 AW971201 AA136350 AA077465

[illegible]

Table 15A lists about 69 genes up-regulated in ovarian cancer compared to normal adult tissues that are likely to be extracellular or cell-surface proteins. These were selected as for Table 14A, except that the ratio was greater than or equal to 3.0, and the predicted protein contained a structural domain that is indicative of extracellular localization (e.g., Ig, FN3, and 7tm domains). Predicted protein domains are noted.

Play	Ex. Acn	UGID	Title	Prot. Dom.	ratio
415589	NA_005760	Hs.111128	ESTs	TM	42.7
428579	NA_005756	Hs.184942	G protein-coupled receptor 64	TM	30.5
428153	AW531043	Hs.184942	similar to GRY-16 containing gene 17	TM	30.1
433982	AS018205	Hs.3378	protein 1, 14-pound) secreted muscle	ES	30.1
433983	AS018205	Hs.3378	secreted, type X, alpha 1 (Secret m	ES	29.2
433984	C11417	Hs.3378	ES	ES	29.2
418007	M13520	Hs.83169	Matrix metalloproteinase 1 (matricu	TM	20.8
409232	AB203137	Hs.7742	Matrix metalloproteinase 1 (matricu	SS_Peptidase_M10	20.6
424098	A351010	Hs.102267	kerl cadherin	TM	17.7
NA_002937	NA_153704	NMA	(never in minis gene 8-related lin	TM	17.4
427356	AW023482	Hs.87849	ESTs	TM	17.4
427355	AJ045672	Hs.288593	EST	TM	17.1
427469	AJ040084	Hs.293347	ESTs	TM	17.0
439993	AA205995	Hs.102267	reagin, beta 8	SS_Thyrog_B	16.7
421155	Hs.87879	Hs.102267	Perf cadherin	ES	16.7
429876	AA205995	Hs.102267	Perf cadherin	ES	15.9
430764	AJ103764	Hs.184955	not binding gene family, member 1	ES	15.9
432978	AA239738	Hs.78978	HA021010 (cancer cell adhesion, aspe	TM	15.1
415299	AA239738	Hs.78978	HA021010 (cancer cell adhesion, aspe	TM	15.0

TABLE 15A: ABOUT 499 UP-REGULATED GENES ENCODING EXTRACELLULAR CELL SURFACE PROTEINS, OVARIAN CANCER VERSUS NORMAL ADULT TISSUES

[illegible]

[illegible]

[illegible]

TABLE 17C:
Play: Unique number corresponding to an Eco probe
Ref.: Sequence source. The 7 digit numbers in this column are Genbank Identifier (GI) numbers. Dunham I. et al.* refers to the publication entitled 'The DNA sequence of human chromosome 22' Dunham, et al. (1995) *Nature* 402:469-495

Play	Ref.	Stund	N_positon
40682	794777	Mens	-110431-110708
40682	794777	Fems	8174-20294
40331	943267	Mens	25359-25776

TABLE 1B: ABOUT 178 UP-REGULATED GENES ENCODING SECRETED PROTEINS, OVARIAN CANCER VERSUS NORMAL ADULT TISSUES

[illegible]

[illegible]

45022	A052235	Ha.52710	EST1, highly similar to ITM1_H	SS,TM,trans	phases,MA1,UBA,SS
45242	NL040654	Ha.157169	ELMO, mod1 kinase	EST1	
45384	A043469	Ha.18491	EST1		
41037	AW17222	Ha.53942	DNF264A157 protein	SS	SS,homo,UPP1,MD,DUF3
44362	AV02733	Ha.31149	serine protease inhibitor, Xan	EST1	SS,TM,velg,CLC,OB
44323	A042733	Ha.13149	EST1		homo,EP1,US,TM,trans
41264	T7141	Ha.22683	EST1, Weakly similar to US3714	EST1	phases
40733	A041565	Ha.20013	homo sapiens cDNA FLJ13179.1a	EST1	SS,trans,trans,trans,trans,trans
45643	AW15147	Ha.86070	cytochrome P450, subfamily 1a	EST1	SS,trans,trans,trans,trans,trans
41160	R3474	Ha.155524	phbHMO103.1 Scara1 placenta	SS	
45585	AW27912	Ha.132070	phbHMO103.9-241195-002-CD3 H	SS	
44387	AW02689	Ha.66219	Formosan synthetase	SS	SS,trans,trans,trans,trans,trans
45394	A444212	Ha.33304	adults center family 2 (fcd)	SS	SS,trans,trans,trans,trans,trans
41912	A420422	Ha.65147	EST1	SS	SS,trans,trans,trans,trans,trans
40801	A516138	Ha.261292	Target Exon	SS	
42388	Ha.261292	Ha.261292	Target Exon	SS	
41710	Ha.191705	Ha.191705	Target Exon	SS	
45065	A024965	Ha.191705	Target Exon	SS	
45902	A024965	Ha.191705	Target Exon	SS	
42201	NL101505	Ha.113207	Grain-like-coupled nucleic acid	SS	SS,TM,trans,trans,trans,trans,trans
40579	AA10248	Ha.278514	COO2599-4501185-0197_00	SS	SS,TM,trans,trans,trans,trans,trans
40149	AW81813	Ha.302740	Epithelial cadherin channel 2	SS	SS,TM,trans,trans,trans,trans,trans
42183	Ha.103354	Ha.103354	Epithelial cadherin channel 2	SS	SS,TM,trans,trans,trans,trans,trans
45819	A011657	Ha.13249	KIA0755 protein	SS	SS,TM,trans,trans,trans,trans,trans
42562	A011655	Ha.71132	homo sapiens, clone IMAGE3181	SS	SS,TM,trans,trans,trans,trans,trans
41289	AW85967	Ha.170152	KIA0755 protein	SS	SS,TM,trans,trans,trans,trans,trans
41105	AW37810	Ha.11123	homo sapiens, clone IMAGE3181	SS	SS,TM,trans,trans,trans,trans,trans
42393	A024965	Ha.191705	Grain-like-coupled nucleic acid	SS	SS,TM,trans,trans,trans,trans,trans
42003	A458293	Ha.111364	EST1, Weakly similar to abcd1	SS	SS,TM,trans,trans,trans,trans,trans
41418	A024965	Ha.191705	Grain-like-coupled nucleic acid	SS	SS,TM,trans,trans,trans,trans,trans
42098	DW142	Ha.241183	homo sapiens, clone IMAGE3181	SS	SS,TM,trans,trans,trans,trans,trans
42362	A024965	Ha.191705	Grain-like-coupled nucleic acid	SS	SS,TM,trans,trans,trans,trans,trans
43843	NL102212	Ha.5215	homo sapiens, clone IMAGE3181	SS	SS,TM,trans,trans,trans,trans,trans
40116	AA16530	Ha.119400	Target Exon	SS	
40106	BE549179	Ha.29008	Target Exon	SS	
41491	BE549179	Ha.29008	Target Exon	SS	
40658	AW92646	Ha.142221	Target Exon	SS	
45035	US138	Ha.6453	Target Exon	SS	
45035	US138	Ha.6453	Target Exon	SS	
40364	NL101198	Ha.311569	Target Exon	SS	
40364	BE14172	Ha.271923	Target Exon	SS	
41794	AW03003	Ha.271923	Target Exon	SS	
433211	AW03003	Ha.271923	Target Exon	SS	
432828	AW03003	Ha.271923	Target Exon	SS	
446100	AW03003	Ha.271923	Target Exon	SS	
42146	R52599	Ha.159714	Target Exon	SS	
42541	A014799	Ha.255005	Target Exon	SS	
42784	A014799	Ha.255005	Target Exon	SS	
42584	A014799	Ha.255005	Target Exon	SS	
42584	A014799	Ha.255005	Target Exon	SS	
41925	US0449	Ha.70673	Target Exon	SS	
413452	A024571	Ha.52883	Target Exon	SS	
421273	A024571	Ha.52883	Target Exon	SS	
432746	A024571	Ha.52883	Target Exon	SS	
432746	A024571	Ha.52883	Target Exon	SS	
40430	AA45229	Ha.24301	Target Exon	SS	
427339	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
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42388	AW14106	Ha.37784	Target Exon	SS	
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42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
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42388	AW14106	Ha.37784	Target Exon	SS	
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42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
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42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.37784	Target Exon	SS	
42388	AW14106	Ha.37784	Target Exon	SS	
41159	AW14106	Ha.3778			

4179	4180	4181	4182	4183	4184	4185	4186	4187	4188	4189	4190	4191	4192	4193	4194	4195	4196	4197	4198	4199	4200	4201	4202	4203	4204	4205	4206	4207	4208	4209	4210	4211	4212	4213	4214	4215	4216	4217	4218	4219	4220	4221	4222	4223	4224	4225	4226	4227	4228	4229	4230	4231	4232	4233	4234	4235	4236	4237	4238	4239	4240	4241	4242	4243	4244	4245	4246	4247	4248	4249	4250	4251	4252	4253	4254	4255	4256	4257	4258	4259	4260	4261	4262	4263	4264	4265	4266	4267	4268	4269	4270	4271	4272	4273	4274	4275	4276	4277	4278	4279	4280	4281	4282	4283	4284	4285	4286	4287	4288	4289	4290	4291	4292	4293	4294	4295	4296	4297	4298	4299	4300	4301	4302	4303	4304	4305	4306	4307	4308	4309	4310	4311	4312	4313	4314	4315	4316	4317	4318	4319	4320	4321	4322	4323	4324	4325	4326	4327	4328	4329	4330	4331	4332	4333	4334	4335	4336	4337	4338	4339	4340	4341	4342	4343	4344	4345	4346	4347	4348	4349	4350	4351	4352	4353	4354	4355	4356	4357	4358	4359	4360	4361	4362	4363	4364	4365	4366	4367	4368	4369	4370	4371	4372	4373	4374	4375	4376	4377	4378	4379	4380	4381	4382	4383	4384	4385	4386	4387	4388	4389	4390	4391	4392	4393	4394	4395	4396	4397	4398	4399	4400	4401	4402	4403	4404	4405	4406	4407	4408	4409	4410	4411	4412	4413	4414	4415	4416	4417	4418	4419	4420	4421	4422	4423	4424	4425	4426	4427	4428	4429	4430	4431	4432	4433	4434	4435	4436	4437	4438	4439	4440	4441	4442	4443	4444	4445	4446	4447	4448	4449	4450	4451	4452	4453	4454	4455	4456	4457	4458	4459	4460	4461	4462	4463	4464	4465	4466	4467	4468	4469	4470	4471	4472	4473	4474	4475	4476	4477	4478	4479	4480	4481	4482	4483	4484	4485	4486	4487	4488	4489	4490	4491	4492	4493	4494	4495	4496	4497	4498	4499	4500	4501	4502	4503	4504	4505	4506	4507	4508	4509	4510	4511	4512	4513	4514	4515	4516	4517	4518	4519	4520	4521	4522	4523	4524	4525	4526	4527	4528	4529	4530	4531	4532	4533	4534	4535	4536	4537	4538	4539	4540	4541	4542	4543	4544	4545	4546	4547	4548	4549	4550	4551	4552	4553	4554	4555	4556	4557	4558	4559	4560	4561	4562	4563	4564	4565	4566	4567	4568	4569	4570	4571	4572	4573	4574	4575	4576	4577	4578	4579	4580	4581	4582	4583	4584	4585	4586	4587	4588	4589	4590	4591	4592	4593	4594	4595	4596	4597	4598	4599	4600	4601	4602	4603	4604	4605	4606	4607	4608	4609	4610	4611	4612	4613	4614	4615	4616	4617	4618	4619	4620	4621	4622	4623	4624	4625	4626	4627	4628	4629	4630	4631	4632	4633	4634	4635	4636	4637	4638	4639	4640	4641	4642	4643	4644	4645	4646	4647	4648	4649	4650	4651	4652	4653	4654	4655	4656	4657	4658	4659	4660	4661	4662	4663	4664	4665	4666	4667	4668	4669	4670	4671	4672	4673	4674	4675	4676	4677	4678	4679	4680	4681	4682	4683	4684	4685	4686	4687	4688	4689	4690	4691	4692	4693	4694	4695	4696	4697	4698	4699	4700	4701	4702	4703	4704	4705	4706	4707	4708	4709	4710	4711	4712	4713	4714	4715	4716	4717	4718	4719	4720	4721	4722	4723	4724	4725	4726	4727	4728	4729	4730	4731	4732	4733	4734	4735	4736	4737	4738	4739	4740	4741	4742	4743	4744	4745	4746	4747	4748	4749	4750	4751	4752	4753	4754	4755	4756	4757	4758	4759	4760	4761	4762	4763	4764	4765	4766	4767	4768	4769	4770	4771	4772	4773	4774	4775	4776	4777	4778	4779	4780	4781	4782	4783	4784	4785	4786	4787	4788	4789	4790	4791	4792	4793	4794	4795	4796	4797	4798	4799	4800	4801	4802	4803	4804	4805	4806	4807	4808	4809	4810	4811	4812	4813	4814	4815	4816	4817	4818	4819	4820	4821	4822	4823	4824	4825	4826	4827	4828	4829	4830	4831	4832	4833	4834	4835	4836	4837	4838	4839	4840	4841	4842	4843	4844	4845	4846	4847	4848	4849	4850	4851	4852	4853	4854	4855	4856	4857	4858	4859	4860	4861	4862	4863	4864	4865	4866	4867	4868	4869	4870	4871	4872	4873	4874	4875	4876	4877	4878	4879	4880	4881	4882	4883	4884	4885	4886	4887	4888	4889	4890	4891	4892	4893	4894	4895	4896	4897	4898	4899	4900	4901	4902	4903	4904	4905	4906	4907	4908	4909	4910	4911	4912	4913	4914	4915	4916	4917	4918	4919	4920	4921	4922	4923	4924	4925	4926	4927	4928	4929	4930	4931	4932	4933	4934	4935	4936	4937	4938	4939	4940	4941	4942	4943	4944	4945	4946	4947	4948	4949	4950	4951	4952	4953	4954	4955	4956	4957	4958	4959	4960	4961	4962	4963	4964	4965	4966	4967	4968	4969	4970	4971	4972	4973	4974	4975	4976	4977	4978	4979	4980	4981	4982	4983	4984	4985	4986	4987	4988	4989	4990	4991	4992	4993	4994	4995	4996	4997	4998	4999	5000	5001	5002	5003	5004	5005	5006	5007	5008	5009	5010	5011	5012	5013	5014	5015	5016	5017	5018	5019	5020	5021	5022	5023	5024	5025	5026	5027	5028	5029	5030	5031	5032	5033	5034	5035	5036	5037	5038	5039	5040	5041	5042	5043	5044	5045	5046	5047	5048	5049	5050	5051	5052	5053	5054	5055	5056	5057	5058	5059	5060	5061	5062	5063	5064	5065	5066	5067	5068	5069	5070	5071	5072	5073	5074	5075	5076	5077	5078	5079	5080	5081	5082	5083	5084	5085	5086	5087	5088	5089	5090	5091	5092	5093	5094	5095	5096	5097	5098	5099	5100	5101	5102	5103	5104	5105	5106	5107	5108	5109	5110	5111	5112	5113	5114	5115	5116	5117	5118	5119	5120	5121	5122	5123	5124	5125	5126	5127	5128	5129	5130	5131	5132	5133	5134	5135	5136	5137	5138	5139	5140	5141	5142	5143	5144	5145	5146	5147	5148	5149	5150	5151	5152	5153	5154	5155	5156	5157	5158	5159	5160	5161	5162	5163	5164	5165	5166	5167	5168	5169	5170	5171	5172	5173	5174	5175	5176	5177	5178	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[illegible][illegible]

419187	AS55535	Hs.94976	ES1s, Weakly similar to A13539	18.6	A212997	Hs.72222	Hypothetical protein FLJ13459	10.1
419188	U24683	Hs.211914	Immunoglobulin heavy constant	9.5	41916	Hs.72222	Hypothetical protein FLJ22924	10.1
419189	BE24708	Hs.15937	ES1s, Weakly similar to A13539	9.5	41917	Hs.72222	Hypothetical protein FLJ22924	10.1
419190	AD27913	Hs.32548	ES1s, Weakly similar to A13539	9.5	41918	Hs.72222	Hypothetical protein FLJ22924	10.1
419191	ES35028	Hs.32548	Hypothetical protein FLJ22924	9.5	41919	Hs.72222	Hypothetical protein FLJ22924	10.1
419192	AW07330	Hs.32548	Hypothetical protein FLJ22924	9.5	41920	Hs.72222	Hypothetical protein FLJ22924	10.1
419193	U24683	Hs.32548	Hypothetical protein FLJ22924	9.5	41921	Hs.72222	Hypothetical protein FLJ22924	10.1
419194	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41922	Hs.72222	Hypothetical protein FLJ22924	10.1
419195	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41923	Hs.72222	Hypothetical protein FLJ22924	10.1
419196	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41924	Hs.72222	Hypothetical protein FLJ22924	10.1
419197	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41925	Hs.72222	Hypothetical protein FLJ22924	10.1
419198	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41926	Hs.72222	Hypothetical protein FLJ22924	10.1
419199	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41927	Hs.72222	Hypothetical protein FLJ22924	10.1
419200	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41928	Hs.72222	Hypothetical protein FLJ22924	10.1
419201	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41929	Hs.72222	Hypothetical protein FLJ22924	10.1
419202	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41930	Hs.72222	Hypothetical protein FLJ22924	10.1
419203	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41931	Hs.72222	Hypothetical protein FLJ22924	10.1
419204	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41932	Hs.72222	Hypothetical protein FLJ22924	10.1
419205	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41933	Hs.72222	Hypothetical protein FLJ22924	10.1
419206	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41934	Hs.72222	Hypothetical protein FLJ22924	10.1
419207	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41935	Hs.72222	Hypothetical protein FLJ22924	10.1
419208	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41936	Hs.72222	Hypothetical protein FLJ22924	10.1
419209	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41937	Hs.72222	Hypothetical protein FLJ22924	10.1
419210	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41938	Hs.72222	Hypothetical protein FLJ22924	10.1
419211	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41939	Hs.72222	Hypothetical protein FLJ22924	10.1
419212	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41940	Hs.72222	Hypothetical protein FLJ22924	10.1
419213	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41941	Hs.72222	Hypothetical protein FLJ22924	10.1
419214	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41942	Hs.72222	Hypothetical protein FLJ22924	10.1
419215	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41943	Hs.72222	Hypothetical protein FLJ22924	10.1
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419226	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41954	Hs.72222	Hypothetical protein FLJ22924	10.1
419227	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41955	Hs.72222	Hypothetical protein FLJ22924	10.1
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419230	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41958	Hs.72222	Hypothetical protein FLJ22924	10.1
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419234	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41962	Hs.72222	Hypothetical protein FLJ22924	10.1
419235	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41963	Hs.72222	Hypothetical protein FLJ22924	10.1
419236	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41964	Hs.72222	Hypothetical protein FLJ22924	10.1
419237	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41965	Hs.72222	Hypothetical protein FLJ22924	10.1
419238	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41966	Hs.72222	Hypothetical protein FLJ22924	10.1
419239	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41967	Hs.72222	Hypothetical protein FLJ22924	10.1
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419241	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41969	Hs.72222	Hypothetical protein FLJ22924	10.1
419242	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41970	Hs.72222	Hypothetical protein FLJ22924	10.1
419243	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41971	Hs.72222	Hypothetical protein FLJ22924	10.1
419244	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41972	Hs.72222	Hypothetical protein FLJ22924	10.1
419245	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41973	Hs.72222	Hypothetical protein FLJ22924	10.1
419246	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41974	Hs.72222	Hypothetical protein FLJ22924	10.1
419247	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41975	Hs.72222	Hypothetical protein FLJ22924	10.1
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419249	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41977	Hs.72222	Hypothetical protein FLJ22924	10.1
419250	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41978	Hs.72222	Hypothetical protein FLJ22924	10.1
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419253	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41981	Hs.72222	Hypothetical protein FLJ22924	10.1
419254	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41982	Hs.72222	Hypothetical protein FLJ22924	10.1
419255	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41983	Hs.72222	Hypothetical protein FLJ22924	10.1
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419257	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41985	Hs.72222	Hypothetical protein FLJ22924	10.1
419258	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41986	Hs.72222	Hypothetical protein FLJ22924	10.1
419259	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41987	Hs.72222	Hypothetical protein FLJ22924	10.1
419260	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41988	Hs.72222	Hypothetical protein FLJ22924	10.1
419261	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41989	Hs.72222	Hypothetical protein FLJ22924	10.1
419262	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41990	Hs.72222	Hypothetical protein FLJ22924	10.1
419263	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41991	Hs.72222	Hypothetical protein FLJ22924	10.1
419264	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41992	Hs.72222	Hypothetical protein FLJ22924	10.1
419265	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41993	Hs.72222	Hypothetical protein FLJ22924	10.1
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419267	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41995	Hs.72222	Hypothetical protein FLJ22924	10.1
419268	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41996	Hs.72222	Hypothetical protein FLJ22924	10.1
419269	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41997	Hs.72222	Hypothetical protein FLJ22924	10.1
419270	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	41998	Hs.72222	Hypothetical protein FLJ22924	10.1
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419272	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42000	Hs.72222	Hypothetical protein FLJ22924	10.1
419273	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42001	Hs.72222	Hypothetical protein FLJ22924	10.1
419274	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42002	Hs.72222	Hypothetical protein FLJ22924	10.1
419275	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42003	Hs.72222	Hypothetical protein FLJ22924	10.1
419276	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42004	Hs.72222	Hypothetical protein FLJ22924	10.1
419277	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42005	Hs.72222	Hypothetical protein FLJ22924	10.1
419278	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42006	Hs.72222	Hypothetical protein FLJ22924	10.1
419279	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42007	Hs.72222	Hypothetical protein FLJ22924	10.1
419280	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42008	Hs.72222	Hypothetical protein FLJ22924	10.1
419281	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42009	Hs.72222	Hypothetical protein FLJ22924	10.1
419282	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42010	Hs.72222	Hypothetical protein FLJ22924	10.1
419283	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42011	Hs.72222	Hypothetical protein FLJ22924	10.1
419284	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42012	Hs.72222	Hypothetical protein FLJ22924	10.1
419285	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42013	Hs.72222	Hypothetical protein FLJ22924	10.1
419286	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42014	Hs.72222	Hypothetical protein FLJ22924	10.1
419287	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42015	Hs.72222	Hypothetical protein FLJ22924	10.1
419288	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42016	Hs.72222	Hypothetical protein FLJ22924	10.1
419289	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42017	Hs.72222	Hypothetical protein FLJ22924	10.1
419290	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42018	Hs.72222	Hypothetical protein FLJ22924	10.1
419291	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42019	Hs.72222	Hypothetical protein FLJ22924	10.1
419292	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42020	Hs.72222	Hypothetical protein FLJ22924	10.1
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419294	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42022	Hs.72222	Hypothetical protein FLJ22924	10.1
419295	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42023	Hs.72222	Hypothetical protein FLJ22924	10.1
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419297	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42025	Hs.72222	Hypothetical protein FLJ22924	10.1
419298	AF161254	Hs.10388	Hypothetical protein FLJ22924	9.5	42026	Hs.72222	Hypothetical protein FLJ22924	10.1
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44402	HL13772	HL1102	hypothetical protein G12959	SS.TM.phases	5.5	41639	HL07231	actin related protein 70	SS.TM.phases	5.5	HL33290	SS.TM.phases
44403	HL13773	HL1103	hypothetical protein G12960	SS.TM.phases	5.5	41640	HL07232	actin related protein 71	SS.TM.phases	5.5	HL33291	SS.TM.phases
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44410	HL13780	HL1110	hypothetical protein G12967	SS.TM.phases	5.5	41647	HL07239	actin related protein 78	SS.TM.phases	5.5	HL33298	SS.TM.phases
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44413	HL13783	HL1113	hypothetical protein G12970	SS.TM.phases	5.5	41650	HL07242	actin related protein 81	SS.TM.phases	5.5	HL33301	SS.TM.phases
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44415	HL13785	HL1115	hypothetical protein G12972	SS.TM.phases	5.5	41652	HL07244	actin related protein 83	SS.TM.phases	5.5	HL33303	SS.TM.phases
44416	HL13786	HL1116	hypothetical protein G12973	SS.TM.phases	5.5	41653	HL07245	actin related protein 84	SS.TM.phases	5.5	HL33304	SS.TM.phases
44417	HL13787	HL1117	hypothetical protein G12974	SS.TM.phases	5.5	41654	HL07246	actin related protein 85	SS.TM.phases	5.5	HL33305	SS.TM.phases
44418	HL13788	HL1118	hypothetical protein G12975	SS.TM.phases	5.5	41655	HL07247	actin related protein 86	SS.TM.phases	5.5	HL33306	SS.TM.phases
44419	HL13789	HL1119	hypothetical protein G12976	SS.TM.phases	5.5	41656	HL07248	actin related protein 87	SS.TM.phases	5.5	HL33307	SS.TM.phases
44420	HL13790	HL1120	hypothetical protein G12977	SS.TM.phases	5.5	41657	HL07249	actin related protein 88	SS.TM.phases	5.5	HL33308	SS.TM.phases
44421	HL13791	HL1121	hypothetical protein G12978	SS.TM.phases	5.5	41658	HL07250	actin related protein 89	SS.TM.phases	5.5	HL33309	SS.TM.phases
44422	HL13792	HL1122	hypothetical protein G12979	SS.TM.phases	5.5	41659	HL07251	actin related protein 90	SS.TM.phases	5.5	HL33310	SS.TM.phases
44423	HL13793	HL1123	hypothetical protein G12980	SS.TM.phases	5.5	41660	HL07252	actin related protein 91	SS.TM.phases	5.5	HL33311	SS.TM.phases
44424	HL13794	HL1124	hypothetical protein G12981	SS.TM.phases	5.5	41661	HL07253	actin related protein 92	SS.TM.phases	5.5	HL33312	SS.TM.phases
44425	HL13795	HL1125	hypothetical protein G12982	SS.TM.phases	5.5	41662	HL07254	actin related protein 93	SS.TM.phases	5.5	HL33313	SS.TM.phases
44426	HL13796	HL1126	hypothetical protein G12983	SS.TM.phases	5.5	41663	HL07255	actin related protein 94	SS.TM.phases	5.5	HL33314	SS.TM.phases
44427	HL13797	HL1127	hypothetical protein G12984	SS.TM.phases	5.5	41664	HL07256	actin related protein 95	SS.TM.phases	5.5	HL33315	SS.TM.phases
44428	HL13798	HL1128	hypothetical protein G12985	SS.TM.phases	5.5	41665	HL07257	actin related protein 96	SS.TM.phases	5.5	HL33316	SS.TM.phases
44429	HL13799	HL1129	hypothetical protein G12986	SS.TM.phases	5.5	41666	HL07258	actin related protein 97	SS.TM.phases	5.5	HL33317	SS.TM.phases
44430	HL13800	HL1130	hypothetical protein G12987	SS.TM.phases	5.5	41667	HL07259	actin related protein 98	SS.TM.phases	5.5	HL33318	SS.TM.phases
44431	HL13801	HL1131	hypothetical protein G12988	SS.TM.phases	5.5	41668	HL07260	actin related protein 99	SS.TM.phases	5.5	HL33319	SS.TM.phases
44432	HL13802	HL1132	hypothetical protein G12989	SS.TM.phases	5.5	41669	HL07261	actin related protein 100	SS.TM.phases	5.5	HL33320	SS.TM.phases
44433	HL13803	HL1133	hypothetical protein G12990	SS.TM.phases	5.5	41670	HL07262	actin related protein 101	SS.TM.phases	5.5	HL33321	SS.TM.phases
44434	HL13804	HL1134	hypothetical protein G12991	SS.TM.phases	5.5	41671	HL07263	actin related protein 102	SS.TM.phases	5.5	HL33322	SS.TM.phases
44435	HL13805	HL1135	hypothetical protein G12992	SS.TM.phases	5.5	41672	HL07264	actin related protein 103	SS.TM.phases	5.5	HL33323	SS.TM.phases
44436	HL13806	HL1136	hypothetical protein G12993	SS.TM.phases	5.5	41673	HL07265	actin related protein 104	SS.TM.phases	5.5	HL33324	SS.TM.phases
44437	HL13807	HL1137	hypothetical protein G12994	SS.TM.phases	5.5	41674	HL07266	actin related protein 105	SS.TM.phases	5.5	HL33325	SS.TM.phases
44438	HL13808	HL1138	hypothetical protein G12995	SS.TM.phases	5.5	41675	HL07267	actin related protein 106	SS.TM.phases	5.5	HL33326	SS.TM.phases
44439	HL13809	HL1139	hypothetical protein G12996	SS.TM.phases	5.5	41676	HL07268	actin related protein 107	SS.TM.phases	5.5	HL33327	SS.TM.phases
44440	HL13810	HL1140	hypothetical protein G12997	SS.TM.phases	5.5	41677	HL07269	actin related protein 108	SS.TM.phases	5.5	HL33328	SS.TM.phases
44441	HL13811	HL1141	hypothetical protein G12998	SS.TM.phases	5.5	41678	HL07270	actin related protein 109	SS.TM.phases	5.5	HL33329	SS.TM.phases
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[illegible]

[illegible]

[illegible]

[illegible]

47845	AW323156	Ha_181022	hypothetical protein FL10038	Collagen
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47908	AW323219	Ha_181085	hypothetical protein FL10101	Collagen
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[illegible]

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[illegible]

[illegible]

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44815	AA15153	Hs.127	anti-infective, delta, deloid	SS,ALAD	21	SEQ ID NO: 1-2
44820	AA17718	Hs.13581	hydrolytic protein FL1032	SS,TM,Sma,PSI,lg	21	SEQ ID NO: 3-4
44825	AA43955	Hs.13581	hydrolytic protein FL1032	Burhopen, C,NA,SS	21	SEQ ID NO: 5-6
44830	AA43955	Hs.13581	hydrolytic protein FL1032	SS,TM,PH22,Chaudh,SS,TM	21	SEQ ID NO: 7-8
44835	AA43955	Hs.13581	hydrolytic protein FL1032	EGF,TLL,SS	21	SEQ ID NO: 9-10
44840	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 11-12
44845	AA43955	Hs.13581	hydrolytic protein FL1032	SS,TM	21	SEQ ID NO: 13-14
44850	AA43955	Hs.13581	hydrolytic protein FL1032	SS,TM,phospho	21	SEQ ID NO: 15-16
44855	AA43955	Hs.13581	hydrolytic protein FL1032	SS,ATP,4PL,C	21	SEQ ID NO: 17-18
44860	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 19-20
44865	AA43955	Hs.13581	hydrolytic protein FL1032	24-CH2C,proth,head,DIL	21	SEQ ID NO: 21-22
44870	AA43955	Hs.13581	hydrolytic protein FL1032	SS,JACOPT,ash,M,ncpt	21	SEQ ID NO: 23-24
44875	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 25-26
44880	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 27-28
44885	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 29-30
44890	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 31-32
44895	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 33-34
44900	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 35-36
44905	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 37-38
44910	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 39-40
44915	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 41-42
44920	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 43-44
44925	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 45-46
44930	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 47-48
44935	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 49-50
44940	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 51-52
44945	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 53-54
44950	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 55-56
44955	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 57-58
44960	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 59-60
44965	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 61-62
44970	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 63-64
44975	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 65-66
44980	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 67-68
44985	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 69-70
44990	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 71-72
44995	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 73-74
45000	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 75-76
45005	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 77-78
45010	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 79-80
45015	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 81-82
45020	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 83-84
45025	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 85-86
45030	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 87-88
45035	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 89-90
45040	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 91-92
45045	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 93-94
45050	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 95-96
45055	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 97-98
45060	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 99-100
45065	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 101-102
45070	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 103-104
45075	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 105-106
45080	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 107-108
45085	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 109-110
45090	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 111-112
45095	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 113-114
45100	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 115-116
45105	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 117-118
45110	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 119-120
45115	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 121-122
45120	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 123-124
45125	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 125-126
45130	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 127-128
45135	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 129-130
45140	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 131-132
45145	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 133-134
45150	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 135-136
45155	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 137-138
45160	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 139-140
45165	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 141-142
45170	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 143-144
45175	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 145-146
45180	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 147-148
45185	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 149-150
45190	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 151-152
45195	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 153-154
45200	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 155-156
45205	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 157-158
45210	AA43955	Hs.13581	hydrolytic protein FL1032	SS	21	SEQ ID NO: 159-160

[illegible]

TABLE 25C.		Ref. Unkno number corresponding to an Eos proteol		Ref. Sequence source. The 7 digit number in this column are GenBank Identifier (G) numbers. "Dinhnam, et al." file	
5		40977		human chromosome 22 Dinham, et al. (1999) JN053 402-489-495	
0		40840		Strand: Indicates DNA strand from which exon were predicted	
15		40840		N-Position: Indicates nucleotide positions of predicted exons	
20		40977		N-Position	
25		40840		Strand	
30		40977		N-Position	
35		40840		Strand	
40		40977		N-Position	
45		40840		Strand	
50		40977		N-Position	
55		40840		Strand	
60		40977		N-Position	
65		40840		Strand	
70		40977		N-Position	
75		40840		Strand	
80		40977		N-Position	
85		40840		Strand	

88

Seq ID NO: 12 Protein sequence

277

Seq ID NO: 14 Protein sequence

Seq ID NO: 15 DNA sequence

279

[illegible]

281

5

10

Seq. ID NO. 34. Protein sequence:
Protein Accession # : A010033.1

Seq ID	NO. 35 DNA sequence	Positive Acid Accretion (n) 1, nH_05893.2	Reading sequence (n:15,85)
40	TGCGCATCTC	CGTCTGTCTGT	GAGTCGCGCA
120	CCCTTGATCC	GATCTATACCA	GACGATATCC
130	CTTGTGGACAC	CCCTGCTCCG	TTCTTCGAGG
140	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
150	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
160	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
170	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
180	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
190	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
200	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
210	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
220	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
230	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
240	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
250	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
260	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
270	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
280	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
290	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
300	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
310	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
320	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
330	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
340	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
350	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
360	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
370	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
380	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
390	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
400	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
410	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
420	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
430	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
440	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
450	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
460	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
470	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
480	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
490	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
500	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
510	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
520	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
530	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
540	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
550	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
560	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
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610	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
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770	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
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790	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
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810	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
820	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
830	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
840	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
850	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
860	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
870	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
880	CCCTTGGACAG	CCCTGCTGCG	AAAGACAGCG
890	CCCTTGGACAG	CCCTGCTGCG	

Seq. in NO. 34 Protein sequence
Protein Accession #. WP 05814.1

1	11	21	31	41		
MA1PTAPLL	GEQTPALGS	LKFLPFLGS	VOSESLGS	TOEASGLS	VLAHPFLGS	491
LLPFLPPLGS	CAVSLQGLTS	RKRLVLA	LR	LP	LP	1201
DLPFLPPLGS	FQDQGLTS	FRFTVAAG	Q	LP	LP	1201
DVPLKALG	DAIRVPLGS	AVSLVPLGS	CPVPLQGLS	Q	LP	1201
ADPPLKALG	DAIRVPLGS	AVSLVPLGS	CPVPLQGLS	Q	LP	1201
ACDQVPLGS	ISDRLVPLGS	MEGLVPLGS	LAATVQGLS	LP	LP	1201
ACDQVPLGS	ISDRLVPLGS	MEGLVPLGS	LAATVQGLS	LP	LP	1201

PQQVPSBVIG HLOZFLQJG PEDIRKNVY SLSTLKALLS WQHQKSPQ VATLIDRPVK 420
 GQOQLOKNTL DTUJATFOY LCBLSGSELS SVPPESINAV RPOLOJOTCP RQLZVLTPQA 480
 RLAPQPMKGS ETPVKIOGFL GADPTEOLKA LSCQVNSQIL ATPMLKRTDA VLPJLVABQ 540
 KLIAPHPVEZ KAEHRRPVR DMWLRQKQDD LZTLQJLQGG QIPNOTVLID LSVQKLSQST 600
 PCJLQGPQVL TVLQMLVAST LA

[illegible]

Seq ID NO.	18	Protein sequences
1	21	Protein Accession # U019756.1
2	22	
3	23	
4	24	
5	25	
6	26	
7	27	
8	28	
9	29	
10	30	
11	31	
12	32	
13	33	
14	34	
15	35	
16	36	
17	37	
18	38	
19	39	
20	40	
21	41	
22	42	
23	43	
24	44	
25	45	
26	46	
27	47	
28	48	
29	49	
30	50	
31	51	
32	52	
33	53	
34	54	
35	55	
36	56	
37	57	
38	58	
39	59	
40	60	

[illegible]

1	TCCTTGATCC	GCACCAAGTG	GCACCAAGTG	TGCGATGTTT	CGAGATATGG	TCCTTGATCC	2101
2	TCTTGATG	GGCCCTGATG	GACCTGACGC	GCATGATCG	CTCTTGACGC	TCCTTGATCC	2102
3	AATCAAGCA	CGCCCTCTATG	CGCTCTATG	TGATCGACAC	GCCTTGATGCG	GAGATGATCG	2220
4	AGGCGCTGAC	CAGCGCTGCG	CGCTTACAGA	TGATCGACAC	GATCTTGATGCG	TGCTTGATGCG	2280
5	CGCTTGATG	CTGATCTGAT	CGCTTGATG	CTGATCTGAT	CTGATCTGAT	TCCTTGATGCG	2340
6	AAGCGATAGCG	CGACCGGAGCG	CGACCGGAGCG	CTAGATGAGCA	AGCGCTTGAC	TCCTTGATGCG	2400
7	CGCTTGATG	CGAGAAAGCG	CGACCTCTGAG	AGAGATGATCG	CGCTTGATGCG	TCCTTGATGCG	2460
8	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	AGAGATGATCG	AGAGATGATCG	TCCTTGATGCG	2520
9	TCCTTGATG	TAGCTGATCG	CGCTTGATG	CGCTTGATG	CGCTTGATG	TCCTTGATGCG	2580
10	TCCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2640
11	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2700
12	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2760
13	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2820
14	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2880
15	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	2940
16	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3000
17	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3060
18	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3120
19	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3180
20	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3240
21	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3300
22	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3360
23	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3420
24	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3480
25	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3540
26	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3600
27	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3660
28	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3720
29	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3780
30	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3840
31	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3900
32	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	3960
33	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	4020
34	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	4080
35	CGCTTGATG	CGACCGGAGCG	CGACCGGAGCG	TGAGATGATCG	CGCTTGATG	TCCTTGATGCG	4140

Seq ID NO. 60 Protein sequence

Protein Accession #, NP_002812

1 11 21 31 41 51

MAHAGRSFAR PIRPULPVLG LUPILQDGT AVTIPMPES QNQLGRAL LACSTIAPRP 60
VHVRGQSPQ PQRSTERRA QGSEGLFAR QUTQUTLAC DILAQDVTQ CVADDTQTE EARNRANRA 120
INTKEMPP LKGRANSA QUTQUTLAC HIGRPATP QPDQDQTS QPDQDQTS QPDQDQTS 180
RANRQVLRQ QGSEGLFAR QGSEGLFAR QGSEGLFAR PANGSLAQ VQVQVQVQ 240
CTQDQDPR ITTAYULVLA LSGDQVLP RTTATGEEER RQDVTPLP SPQWNNHAG 300
VALPQTHRVY ITTAYULVLA LSGDQVLP QNANLQVLP RQDVTPLP VSNLAKQD 360
SQSEKTPSI LQCLTQATP PTVYTNVLR LIGSDQVLP FVQCTALAS VFTDVTWRA 420
QSGSTFAPSI LQCLTQATP KLKTFPPQD QGQDQDEFA TPTQSTQNS KPTTNNEDRA 480
GGSLPYNVTD HANGFLYAR TDADRQGTQ IASNGTQGI RARVQLYAR FTTTTPVPR 540
TPTVQDRTAL LAGCAQDQD PLIQGVQDR ILOFTPLGR MHTFQVSLV IADVAPEDSG 600
RTTCLQDRCR HIKRTAPLYR VQDVEPESL QGQDQDPRP HQTQDQVLP AQANVATVIL 660
GLATPQDRCR KAPLQDQD QGSEGLFAR QGQDQDPRP QGQDQDPRP QGQDQDPRP 720
NKHSTEDER PRSLQUTP ITLQSGSPER PANGSLAQ QVATVULV LQATQDQD 780
QVATVULV LQATQDQD QGSEGLFAR QGSEGLFAR QGSEGLFAR QGSEGLFAR 840
PLSTQUTAL CTQVQVQVQ LSGHSTEDER LANKVLA QVQVULVLA LQATQDQD 900
YHPLQDPRP RQKSPSLR QPSTQDQDPRP QPSTQDQDPRP QPSTQDQDPRP QPSTQDQDPRP 960
ACGASLQDPRP QPSTQDQDPRP QPSTQDQDPRP QPSTQDQDPRP QPSTQDQDPRP QPSTQDQDPRP 1020

[illegible]

Seq ID NO: 43 Protein sequence
Protein Accession #: NP_006094

1 11 21 31 41 51
MPACRGLPFA MALLGELLG OPTLVSTGA ETATGVCFSA AGQVCTQCYA EDSBCDNLK
CEGAAATGAC TACDNDGDS CPYVAILHPPQ LALSDGQWY DSCGPMGIC CRRGCKVSC
VTIHF.

80
Seq ID NO: 43 DNA sequence

85 130

Seq ID NO: 63 DNA sequence

[illegible]

Seq ID NO.	64 Protein sequences	Accession #	1	11	21	31	41	51
40	DELSAPAVG	VTQKEDQSD	TAPTPAPV	RKNCHQPS	DSVNVICST	QVCTHIED		60
	MLGAPVPTGS	QVTEKEDQSD	CDPTTPHQR	RISECTETPN	SCNDVURLT	PLQKQNDPVD		130
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		240
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		360
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		480
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		600
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		720
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		840
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		960
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		1080
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		1200
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		1320
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		1440
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		1560
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		1680
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		1800
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		1920
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		2040
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		2160
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		2280
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		2400
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		2520
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		2640
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		2760
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		2880
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		3000
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		3120
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		3240
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		3360
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		3480
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		3600
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		3720
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		3840
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		3960
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		4080
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		4200
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		4320
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		4440
	QVTHIDALLI	SVYLLALVI	LIUUFQYPI	KQKSTPEVPS	ICQKQETPL	PGKSTJADLI		4560
	ESQSGESG	GVPLQVALL	ALQYQVQVI	QKSTQYQVM	GMKGVKAV	KVFPTTAS		4680
	MPKSTIQT	VAKHSHIT	FIANDKQAN	SDQNTLTD	YHGGKGLTD	LAETTTKDN		4800
	TKALATYS	GLQKLEHTI	STQKQKATN	IKLQKSHLV	NKQKCTCQ	LAGAVVPS		

Seq ID NO: 65 DNA sequence
Nucleic Acid Accession #: NM_006475.1
Coding sequence: 28..2518

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[illegible][illegible][illegible][illegible]

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Seq. ID. NO. 84 DNA sequence									
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Coding sequence: 89-2356									
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TGCTGCTG	TCTCTTGAT	ATATACAG	CCCTCTCTTC	TCTCTCTG	ATATACCTTG	120			
TGTTTCTTG	TCTTTCAGG	TGCTCCCTAT	CCAGAGCTTG	TCTCTCTG	ATATACGCA	180			
AGACACTC	GGAGAGATT	CAGCTCCG	CAATATATG	TCTCTCTG	CTCTGTGGA	240			
AGCTGCTG	ATCATCTTG	GGATCTTG	GCATCTTG	TCTCTCTG	CTCTGTGGA	300			
ACGCTGCG	TTCATGCTA	CTCTGCTAT	GCATCTTG	TTCATGCTA	TTCATGCTA	360			
ACACAGATG	AAGGACAGA	TACAGAGAT	GATTTTCAA	GGAGAGCTCA	GGAGAGCTCA	420			
CTATCTCTG	ATATATCTTC	TGCACAGAG	GTCTCCAGG	ATATATGCTA	TGCTCTTGCC	480			
CCAACTCTG	TAATCTGTA	GTCCCTGGAG	ATATATGCTA	GTCCCTGCTA	TGCTCTTGCC	540			
CGACTAGAT	GAAGAGGCTG	TGACTGTTT	TGCTGTGGGG	GTCTCTGTTT	GTCTCTGTTT	600			
CGACTAGAT	GCATCTGAT	AGGCGCTGAT	GTCTCTGTTT	GTCTCTGTTT	GTCTCTGTTT	660			

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Seq ID NO: 95 Protein sequence
Protein Accession #: XP_051860.2

307

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Seq ID NO: 96 DNA sequence
Nucleic Acid Accession #: NM_020436 and AK001666
Coding sequence: 63-3224

308

Seq ID NO: 102 DNA sequence
Nucleic Acid Accession #: AK025790

[illegible]

Seq ID NO. 103 Protein sequence
Protein Accession # U03374.1

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SGKQVILPA	VLEPDSVRL	RQDQCVRI	ENVEVLQA	PROFALKSN	REGIQVQTR
120	130	140	150	160	170
PRFALPNS	VQGLSPFTD	VIEKVVQRI	QOMGLITVQ	VTHSQTITL	QOTIEQDIL
180	190	200	210	220	230
PRFALPNS	LOQGLSPFTD	PLKLVKSVRI	WDSQGLTVE	KHKLQSLAG	GLAESELETS
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300	310	320	330	340	350
LVKLVKSV	RTITQSPDS	LAGDSQGLT	TSQSQGLTS	HRWKLQSLA	QVAVPYNRS
360	370	380	390	400	410
VKLVKSV	RTITQSPDS	LAGDSQGLT	TSQSQGLTS	HRWKLQSLA	QVAVPYNRS
420	430	440	450	460	470
GVKLVKSV	RTITQSPDS	LAGDSQGLT	TSQSQGLTS	HRWKLQSLA	QVAVPYNRS
480	490	500	510	520	530
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Seq ID NO. 104 DNA sequence

[illegible]

313

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[illegible][illegible]

Seq ID NO: 120 DNA sequence
Nucleic Acid Accession: J1_BK_003377
Coding sequence: 121..1194

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ACGATGATGA	ACTGATGATC	TGACTATATC	TAGCTATATC	AGGCTGAGGA	GAATTTATAC
CGCTTCATCA	CGGACGAGGA	ACGCAATGCG	AGAGATATCG	CTCTGCTGAC	TGCGCGCTTG
ACCTTCGATG	GGGACGAGGA	ACGCAATGCG	CAGCTTTGCT	CTCTGCTGAC	TGCGCGCTTG
GGCTTCCTCA	TGCGGCTGCA	CTGATGATGA	ACTAGGATGA	CAGCTTCGGA	CGCGCTGAGG
AGCGATGATGA	GTATGCTGCG	CGGCTCTGCG	GAATCTGCGC	GAATCTGCGC	AGAGCGATGC
AGCGATGATGA	ACTATGCTGC	CGAGCTGATC	CGCGGCTGAC	CTCTGCTGCT	CTTCTGCTGT
TTGTTGGGCG	CGATGATGCG	TCGAGGTCGC	TCGAGGTCGC	TCGAGGTCGC	TCGAGGTCGC
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AGTATGCTG	AGTATGCTG	TAGCGAGGCA	GAATGCTGCG	GAATGCTGCG	CTCTGCGAGT
CGAGGAGTGG	CAGATGATG	TATGAGTCT	TTTTTCTGCG	TTTTTCTGCG	TCTGCGAGG
CTCTCTCTCC	TAGCTATGCT	GGATGCTGCT	TGCGTCACTA	TGCGTCACTA	AAAGCTGACG
CGACTGATCT	AGAGAGATCT	GGATGCTGCT	TGCGTCACTA	TGCGTCACTA	AAAGCTGACG
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WHAT IS CLAIMED IS:

- 1 1. A method of detecting an ovarian cancer-associated transcript in a cell
- 2 from a patient, the method comprising contacting a biological sample from the patient with a
- 3 polynucleotide that selectively hybridizes to a sequence at least 80% identical to a sequence
- 4 as shown in Tables 1-26.
- 1 2. The method of claim 1, wherein the biological sample comprises
- 2 isolated nucleic acids.
- 1 3. The method of claim 2, wherein the nucleic acids are mRNA.
- 1 4. The method of claim 2, further comprising the step of amplifying
- 2 nucleic acids before the step of contacting the biological sample with the polynucleotide.
- 1 5. The method of claim 1, wherein the polynucleotide comprises a
- 2 sequence as shown in Tables 1-26.
- 1 6. The method of claim 1, wherein the polynucleotide is immobilized on
- 2 a solid surface.
- 1 7. The method of claim 1, wherein the patient is undergoing a therapeutic
- 2 regimen to treat ovarian cancer.
- 1 8. The method of claim 1, wherein the patient is suspected of having
- 2 ovarian cancer.
- 1 9. An isolated nucleic acid molecule consisting of a polynucleotide
- 2 sequence as shown in Tables 1-26.
- 1 10. The nucleic acid molecule of claim 9, which is labeled.
- 1 11. An expression vector comprising the nucleic acid of claim 9.
- 1 12. A host cell comprising the expression vector of claim 11.
- 1 13. An isolated polypeptide which is encoded by a nucleic acid molecule
- 2 having polynucleotide sequence as shown in Tables 1-26.

- 1 14. An antibody that specifically binds a polypeptide of claim 13.
- 1 15. The antibody of claim 14, further conjugated to an effector component.
- 1 16. The antibody of claim 15, wherein the effector component is a
- 2 fluorescent label.
- 1 17. The antibody of claim 15, wherein the effector component is a
- 2 radioisotope or a cytotoxic chemical.
- 1 18. The antibody of claim 15, which is an antibody fragment.
- 1 19. The antibody of claim 15, which is a humanized antibody
- 1 20. A method of detecting an ovarian cancer cell in a biological sample
- 2 from a patient, the method comprising contacting the biological sample with an antibody of
- 3 claim 14.
- 1 21. The method of claim 20, wherein the antibody is further conjugated to
- 2 an effector component.
- 1 22. The method of claim 21, wherein the effector component is a
- 2 fluorescent label.
- 1 23. A method for identifying a compound that modulates an ovarian
- 2 cancer-associated polypeptide, the method comprising the steps of:
- 3 (i) contacting the compound with an ovarian cancer-associated polypeptide,
- 4 the polypeptide encoded by a polynucleotide that selectively hybridizes to a sequence at least
- 5 80% identical to a sequence as shown in Tables 1-26; and
- 6 (ii) determining the functional effect of the compound upon the polypeptide.
- 1 24. A drug screening assay comprising the steps of
- 2 (i) administering a test compound to a mammal having ovarian cancer or a cell
- 3 isolated therefrom;
- 4 (ii) comparing the level of gene expression of a polynucleotide that selectively
- 5 hybridizes to a sequence at least 80% identical to a sequence as shown in Tables 1-26 in a

- 6 treated cell or mammal with the level of gene expression of the polynucleotide in a control
- 7 cell or mammal, wherein a test compound that modulates the level of expression of the
- 8 polynucleotide is a candidate for the treatment of ovarian cancer.

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